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ILLUSTRATING DR. WOODS'S ARTICLE ON "OCULAR ANAPHYLAXIS."
III.—THE RÔLE OF UVEAL PIGMENT.

FIG. 1.—Appearance of pigment heated in preparation.

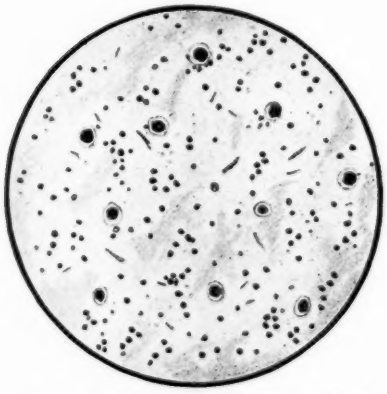

FIG. 2.—Appearance of pigment prepared without heating.

ARCHIVES OF OPHTHALMOLOGY.

OCULAR ANAPHYLAXIS.

III. THE RÔLE OF UVEAL PIGMENT.

BY DR. ALAN C. WOODS, PHILADELPHIA.

(With two illustrations on Text-Plate VII.)

From the John Herr Musser Department of Research Medicine, University of Pennsylvania.

THE anaphylactic theory of sympathetic ophthalmia assumes that the disintegrating uveal tissue in the exciting eye is absorbed and creates a hypersensitiveness of the second eye. A reaction between the antibodies formed in the sympathizing eye, and the exciting uvea, produces an intoxication which is manifested clinically as a sympathetic ophthalmia. The possibility or impossibility of this theory hinges fundamentally upon the antigenic properties of uveal tissue, or, more exactly, upon the ability of uveal tissue to act as a foreign protein in the same species of animal, and thus to produce a sensitization and intoxication. Furthermore, uveal tissue, or the active constituent, must be organ specific and lack species specificity.[1] The relationship of this last point to the anaphylactic theory will be discussed later.

[1] Organ specificity implies that in animals sensitized to a given organ or tissue, anaphylactic intoxication may be produced by reinjection of the same organ or tissue, irrespective of the species of animal from which obtained.

Species specificity implies that in animals sensitized to protein of a given species, anaphylactic intoxication may be produced by reinjection of any protein from an animal of the same species from which the original protein was obtained, with little regard to the organ.

In the previous papers of this series (1, 2) it has been shown that if the eyes of an animal, generally sensitized to a given protein, are perfused with normal defibrinated blood to which the same protein has been added, anaphylactic phenomena result in the eye. These phenomena consist in a marked contraction of the pupil and the occurrence of hemorrhages in the fundi.

This ocular reaction was utilized to determine the antigenic properties of uveal tissue. As a result of numerous perfusion experiments, it was found that uveal tissue could act as antigen in animals of the same species, and could produce anaphylactic phenomena in the eye of a generally sensitized animal, by intoxication through the blood stream. Further, uveal tissue was organ specific, anaphylactic intoxication being produced by perfusion of uvea of another species. At the same time, it was shown that there was considerable reason to believe that the pigment of the uveal tract was the constituent of uveal tissue responsible for these remarkable antigenic properties. However, so many technical difficulties were encountered in the preparation of the pigment that it was impossible to state this last fact with any degree of surety.

Elschnig (3) supposed the pigment to be the constituent of the uvea responsible for its antigenic properties, and claimed to have shown this by the complement fixation reaction. Von Szily (4), however, using general anaphylactic phenomena as his guide, was unable to substantiate Elschnig's work and believed the pigment to be without antigenic properties. If sympathetic ophthalmia is an anaphylactic uveitis, it is of paramount importance, both for the scientific value of the observation, and for the ultimate diagnosis of the disease, to determine the exact tissue responsible for the anaphylactic reaction. Accordingly, using the ocular anaphylactic reaction to perfusion as the method, a study has been made of this question.

PREPARATION OF PIGMENT.

The methods for the preparation of the uveal pigment, used by Elschnig and von Szily, differ slightly. The first step in Elschnig's method consists in preserving the eyes in

1 per cent. formalin. The uveal tract is removed as described in a previous paper (2). The uveæ are then macerated, and heated with a 12 per cent. acid salt in a water bath (boiling) for seven hours. The pigment is removed by centrifuging and is washed free of acid. It is then washed lipoid free with alcohol and ether, and finally dried at 100°.

Von Szily's method consists in macerating the uvea in distilled water. The pigment is gradually freed, and the water-containing pigment poured off, fresh water being added until the uveæ no longer color the water. This water extract of the uveæ is filtered, the pigment passing through. To this pigment-water filtrate is added an equal volume of saturated solution of ammonium sulphate. This is allowed to stand a short time, and is then heated to 80°, at which temperature the pigment coagulates and may be removed by filtration. This pigment is washed free of lipoids with alcohol and ether, and is dried, either by heat or in a vacuum.

We have had no experience with pigment prepared by Elschnig's method. Many preparations of pigment have been made by von Szily's method, and in practically all cases these preparations have been very unsatisfactory. Primarily, there is much reason to believe that pigment so prepared cannot be absorbed by the animal. Such pigment is absolutely insoluble in any ordinary solvent. In suspension, in spite of much previous grinding and prolonged shaking, it quickly settles out as a fine insoluble sediment. Animals injected intraperitoneally with such a suspension show at autopsy the pigment, apparently unabsorbed, still present over the visceral peritoneum. Further, when such pigment preparations were used in the perfusion apparatus, constant thrombosis of the vessels or plugging of the apparatus was encountered, with a consequent summary cessation of perfusion.

The microscopic appearance of such pigment is shown in Figure 1. There are two types of pigment corpuscles, a large and a small, and short needles. The pigment corpuscles are clumped together in large and small masses, which we could find no means to break up.

Consequently we have prepared the pigment in a slightly different manner from the methods given by either Elschnig

or von Szily. The pigment is removed from the uveæ by maceration with distilled water, as recommended by von Szily, and after filtration an equal volume of saturated solution of ammonium sulphate is added. This mixture is not heated, however, but is allowed to stand at room temperature for twenty-four hours, during which time the pigment gathers in a scum at the top of the mixture, and sediments out at the bottom. The pigment is now removed by centrifuging at high speed, and is repeatedly washed with distilled water. It is finally taken up in salt solution so that 7.5*cc* of the resulting suspension shall contain the pigment of one uvea. This emulsion is now shaken in a shaking machine for a few hours. A dead black suspension results which does not sediment upon standing and does not cause thrombosis or plugging when used in the perfusion apparatus. From the evidence at the autopsy of animals injected intraperitoneally with such pigment, it is readily absorbed.

The microscopic appearance of such pigment is shown in Figure 2. The corpuscles are distinct and separate. There is no clumping, the whole suspension is homogeneous. However, if this pigment emulsion is heated to even 80°, it assumes at once the appearance shown in Figure 1 and again appears to be unabsorbable.

The chemical steps taking place in such a preparation are probably as follows. The maceration of the uvea with distilled water and consequent extraction removes not only the pigment corpuscles, but also the serum and other albumens, the globulins, and globins. The addition of the ammonium sulphate precipitates the pigment, globulins, and globins; the albumens, however, stay in solution, and are removed from the pigment when the mixture is centrifuged, and the supernatant fluid pipetted off the pigment-globulin-globin mixture. When this pigment-globulin-globin mass is repeatedly washed with distilled water, the ammonium sulphate is washed out. Since the precipitation of the globulins and globins by ammonium sulphate is a reversible reaction, the washing out of the ammonium sulphate again frees the globulins and globins, and these are accordingly removed with washing. However, the precipitation of the pigment with ammonium sulphate seems to be an irreversible reaction, for

the washing out of the ammonium sulphate does not free the pigment, which is constantly thrown down with each centrifuging. The removal of the globulins and globins leaves the pigment in what should theoretically be a chemically pure condition, barring the presence of lipoids. It is doubtful, however, if the actual separation and purification of the pigment is as complete as it theoretically appears.

The effect of heating the original mixture of pigment-water and ammonium sulphate, as practiced by von Szily, is to coagulate every protein constituent in the mixture, reducing the pigment, globulins, globins, and albumens from a colloidal to a "granular" state, a condition in which further washing can only remove the ammonium sulphate and lipoids.

CHEMICAL NATURE OF THE PIGMENT.

The chemistry of melanins, to which group of pigments uveal pigment belongs, has long been a subject of interest to both chemists and pathologists. The consensus of opinion (5) is that chemically they are protein derivatives, derived possibly from the blood pigments, but more probably from the protein chromogens. Schmiedberg (6) has prepared artificial melanins from protein. The melanins seem to represent a peculiar type of protein, or protein derivative, occurring in both vertebrate and non-vertebrate animals.

Uveal pigment was first studied by Scherer (7) as early as 1841. Since then it has been carefully studied by Rosow (8), Sieber (9), and Landolt (10). It is in general a combination of nitrogen, hydrogen, oxygen, and carbon together with iron and phosphorus. The various analyses of the C, H, N, and O of the several observers are shown in Table I.

TABLE I.

CHEMICAL COMPOSITION OF UVEAL PIGMENT.

Author	*Carbon*	*Hydrogen*	*Nitrogen*	*Oxygen*
Scherer	58.27	5.97		21.98
Sieber	60.34	5.02	10.81	23.83
Rosow	54.29	5.35	10.18	30.18
Landolt	54.56	5.34	12.70	27.40

Landolt, after elaborate chemical studies, concluded that uveal pigment had no relationship to any blood pigment, but was similar to the indol-producing aromatic complexes of the protein molecule.

No comprehensive studies of the chemical nature of the pigment have been made by us, beyond identifying the substance with that prepared and analyzed by other workers.[1] The pigment suspension examined in the spectroscope cuts off slightly the red end of the spectrum. It is readily oxidized by hyperchloride, the resulting oxidation product showing no characteristic spectra. The pigment gives a weak Millon's reaction. The nitrogen content of the preparations used by us was 9.87 per cent., slightly lower than that reported by other observers. We have made no quantitative estimations of the carbon, hydrogen, and oxygen content. The pigment gives a strong qualitative reaction for iron, and a weaker reaction for sulphur and phosphorus. In all respects, in which we have analyzed it, our results are similar to those already reported by other observers.

EXPERIMENTAL.

Having prepared finally a pigment emulsion which is at least relatively pure, is readily absorbed, and which can be utilized in perfusion without causing thrombosis or plugging, we have endeavored to determine by perfusion whether this pigment is the constituent of the uveal tract responsible for the peculiar antigenic properties of uveal tissue. To do this we have first determined whether or not such pigment can act as an antigen. Next we have determined if the pigment can act as antigen in animals of the same species, as has already been shown to be a property of uveal tissue. Lastly we have determined the questions of organ and species specificity of the pigment reactions.

I. ANTIGENIC PROPERTIES OF HETEROLOGOUS (FOREIGN) PIGMENT.

In this experiment dogs were sensitized by intraperitoneal injection of 7*cc* of cow's uveal pigment, prepared as described.

[1] I am indebted to Dr. Harry Dubin, my colleague in the Department of Research Medicine, for the chemical work here reported.

Three weeks later they were perfused with normal defibrinated blood, to which, after preliminary observations had been made, cow's pigment was added. Three dogs were used in this experiment. All gave extreme reactions, the contraction of the pupil in every case being at least to *2mm* in diameter. Hemorrhages were observed in the fundi of all three animals.

The protocol of one dog, 16–87, is given.

Dec. 14. Sensitized by intraperitoneal injection of *7cc* cow's uveal pigment.

Jan. 8. Perfusion begun at 10.45 A.M. Head washed out with *1300cc* normal salt and normal defibrinated blood solution. Perfusion fluid *800cc* defibrinated normal blood, *350cc* salt solution.

10.45 A.M. Both pupils are *10mm* in diameter. Fundi are negative.

10.55 A.M. Pupils *10mm*, fundi normal. *50cc* cow's pigment emulsion added to perfusion fluid.

11 A.M. Right pupil is *4mm* in diameter, left pupil is *7mm*. Fundi are engorged.

11.15 A.M. Right pupil is *2mm* in diameter, left is *4mm*. Fundi unchanged.

11.20 A.M. Both pupils measure *3mm* in diameter.

11.28 A.M. Right pupil now measures *4mm*, left measures *2mm*. Right fundus shows several small areas of reddish haze. What little of left fundus can be seen is negative. Winking reflex gone.

11.42 A.M. Right pupil is *4mm* in diameter. Left pupil is pin-point. Right fundus shows several small hemorrhages. Left fundus not seen.

12.00 M. Pupils are unchanged in size, right being *4mm* and left pin-point. Right fundus shows hemorrhages before noted. Left fundus not seen.

12.15 P.M. Right pupil is *5mm*, left is *3mm*. Right fundus as before. Left fundus shows a blotchy hemorrhage over disk, and a smaller one near upper nasal vein.

12.30 P.M. Right pupil measures *6mm* in diameter, left measures *5mm*. Fundi show the hemorrhages before noted, now slightly larger in size.

These perfusions definitely establish the first point—that foreign pigment can act as antigen, and can produce ocular intoxication via the blood stream in sensitized animals.

The next point is the ability of homologous pigment to act as antigen.

II. ANTIGENIC PROPERTIES OF HOMOLOGOUS (DOG'S) PIGMENT.

To determine this point, dogs were sensitized by intraperitoneal injection of dog's pigment, and were later perfused with normal defibrinated blood to which dog's pigment was added. The dogs perfused in this experiment all reacted to the perfusion with positive results, showing a prompt contraction of the pupil and hemorrhages in the fundi.

The protocol of one of these dogs, 16–48, is given below.

April 12. Sensitized by intraperitoneal injection of 7*cc* of dog's pigment suspension.

May 1. Perfusion started at 11 A.M. Head washed out as usual. Perfusion fluid 600*cc* normal defibrinated blood, 250*cc* salt solution.

11 A.M. Pupils equal 10*mm* in diameter. Fundi appear normal.

11.10 A.M. Pupils are 10*mm*; fundi are negative. 20*cc* dog's pigment suspension added to perfusion fluid.

11.12 A.M. Right pupil is 3*mm* in diameter, left pupil is pin-point. Right fundus appears negative. Left fundus not seen.

11.30 A.M. Right pupil is 2*mm* in diameter. Left pupil is 5*mm*. Both fundi show engorged vessels, and in right fundus there is a small hemorrhage over the disk.

11.40 A.M. Pupils are unchanged in size. The hemorrhage over the right disk is much larger.

11.50 A.M. Both pupils measure 5*mm* in diameter. Both fundi show a general seepage of blood over the lower portions of the fundi, covering two thirds of the right fundus and a half of the left fundus. The pericorneal injection of both eyes is intense.

12.10 P.M. There is no change in the pupils or appearance of the fundi.

12.30 P.M. The pupils each equal 6*mm* in diameter. The lower half of both fundi is covered with a thin film of blood.

Perfusion stopped.

This demonstrates the antigenic property of pigment in the homologous animal, and substantiates Elschnig's contention that it is the pigment which is responsible for the antigenic properties of uveal tissue. To further demonstrate this point, dogs have been sensitized to emulsion of dog's uvea, which contains both the pigment and the other constituents

of the uvea, and subsequently perfused with pigment suspension. These dogs reacted to the perfusion with the typical ocular anaphylactic phenomena, showing that intoxication is dependent upon the pigment. The two dogs perfused showed contraction of the pupil from 10*mm* to 3*mm* and from 11*mm* to 4*mm* in diameter. The protocols are practically similar to those already given.

These two experiments, together with the work previously reported, establish the first and cardinal point assumed in the anaphylactic theory of sympathetic ophthalmia—uveal tissue possesses antigenic properties in the homologous animal, and it is the pigment of the uveal tract that is responsible for the phenomena.

ORGAN SPECIFICITY.

The remaining points concerning the antigenic properties of uveal tissue assumed in the anaphylactic theory are the points of the specificity of uveal tissue. The pigment responsible for the antigenic property of uveal tissue must be organ specific and not species specific. The reasons for this are at once apparent. If the uveal pigment absorbed from the exciting eye produces a hypersensitiveness of the second eye, intoxication in the second eye must be produced by uveal pigment alone, and not by other body protein. If this were not so, and other body protein could produce intoxication, and the enucleation of the exciting eye, which is equivalent to the removal of the exciting pigment, would be of no value while other equally dangerous protein remained behind. If the pigment were not lacking in species specificity, the blood serum, which is the "fluid extract of the organism," would theoretically produce intoxication. To establish a scientific basis for the anaphylactic theory, the pigment must be shown to be organ specific and not species specific.

To demonstrate the organ specificity of the pigment, dogs have been sensitized to one species pigment and subsequently perfused with the pigment of another species. One dog was sensitized to cow's pigment and perfused with dog's pigment, while a second was sensitized to dog's pigment and perfused with cow's pigment. Both dogs reacted positively, showing

upon perfusion with the organ specific antigen, the typical anaphylactic reaction in the eyes. There was no essential difference between the degree of the reaction in the two dogs. The protocol of dog 16–68 is given.

Dec. 14. Sensitized by intraperitoneal injection of cow's pigment suspension.

Jan. 11. Perfusion started at 10.30 A.M. Head washed out as usual. Perfusion fluid 800*cc* normal defibrinated blood. 40*cc.* salt solution.

10.30 A.M. Pupils are 12*mm* in diameter. Fundi appear normal.

10.38 A.M. Pupils are 12*mm*; fundi are normal. 20*cc* dog's pigment suspension added to the perfusion fluid.

10.45 A.M. Both pupils have contracted since the addition of the dog's pigment, and now measure 3*mm* each in diameter. The fundi show no change except for a slight dilation of the vessels.

11.00 A.M. Both pupils have redilated slightly, now measuring about 6*mm* in diameter. The right fundus shows two small areas of reddish haze to either side of the disk. The left fundus shows a similar haze above the disk.

11.15 A.M. The pupils measure 6*mm* in diameter. The areas of reddish haze noted before in the fundi are now well defined hemorrhages.

11.30 A.M. Pupils are 6*mm* in diameter. Fundi as before.

11.45 A.M. Pupils are unchanged in size. The hemorrhages in the fundi are somewhat larger.

12 M. Right pupil is 5*mm* in diameter, left is 7*mm*. Fundi are unchanged.

12.15 P.M. There is no change since the last note.

12.30 P.M. Right pupil measures 5*mm*, left measures 6*mm* in diameter. There are several blotchy hemorrhages in each fundus. Perfusion stopped.

This experiment shows that the pigment is organ specific in its action. Intoxication after sensitization is dependent upon the organ, or tissue—the pigment—irrespective of its species. Hand in hand with the establishment of this, comes the final point to be proved: that after sensitization with pigment, other protein from the same species animal will not produce intoxication—the lack of species specificity.

SPECIES SPECIFICITY.

Using the complement fixation reaction of the sera of uvea and pigment immune rabbits, Elschnig showed that whole uvea is species specific in its reaction, while the pigment fulfills the assumption of the anaphylactic theory of sympathetic ophthalmia, and is not species specific. The reasons for this are apparent. Whole uveal emulsion contains not only the non-species specific pigment, but also other elements, blood, smooth muscle, and connective tissue, which elements are common to all the tissues of the body. These elements react like ordinary body protein, and are strongly species specific. After sensitization with such tissue, if from a different species animal, intoxication may be elicited by reinjection with such tissue from the same species animal. Sensitization, or immunization with emulsion of whole uvea of a different species, gives accordingly a sensitization or immunization to organ-specific pigment, and to species-specific tissue, blood, muscle, etc. Heterologous uveal emulsion presents accordingly the anomaly of being both organ and species specific.

To study more completely the species-specific properties of uveal tissue, dogs have been sensitized to the emulsion of whole uvea and to uveal pigment of the cow and subsequently perfused with indifferent protein of this species. The emulsion of foreign uvea (as shown by Elschnig) was found to be species specific in its reaction. Two dogs were sensitized with cow's uveal emulsion and were subsequently perfused with normal defibrinated dog's blood to which was added cow's serum, liver, and kidney extracts. These dogs reacted positively to such perfusion, giving moderately intense ocular anaphylactic reactions. The protocol of one dog, 16–62, is given.

May 18. Sensitized by intraperitoneal injection of 5*cc* cow's uveal emulsion.

June 1. Perfusion started at 11.30 A.M. Head washed out as usual. Perfusion fluid 800*cc* normal defibrinated blood, 350*cc* salt solution.

11.45 A.M. Pupils equal 9*mm* in diameter. Fundi appear normal. 50*cc* cow's blood serum, 50*cc* 4 per cent. kidney extract, and 50*cc* 4 per cent. cow's liver extract added to the perfusion fluid.

12 M. Pupils are 8*mm* in diameter. Fundi appear normal.

12.30 P.M. Pupils have gradually contracted to 4*mm* each in diameter. Fundi appear normal.

12.45 P.M. Both pupils are 5*mm* in diameter. Fundi are engorged but no hemorrhages are apparent.

1.00 P.M. Right pupil measures 5*mm*, left pupil 3*mm* in diameter. There is one small hemorrhage visible in both fundi.

1.20 P.M. Right pupil is 5*mm*, left pupil 3*mm* in diameter. Fundi show marked engorgement of the vessels and the hemorrhages before noted.

1.40 P.M. Right pupil is 6*mm*, left pupil 4*mm* in diameter. Fundi are as before noted.

2.00 P.M. Right pupil is 7*mm*, left pupil is 4*mm* in diameter. Right fundus shows several hemorrhages, left fundus shows the one hemorrhage before noted.

2.20 P.M. Right pupil is 8*mm*, left pupil 4.5*mm* in diameter. The fundi show no further change. Perfusion stopped.

While, as this experiment shows, dogs sensitized to cow's whole uvea emulsion reacted positively when perfused with other cow's protein, dogs sensitized to cow's pigment showed no reaction when perfused with other protein of the cow. This point is shown by the following experiment. Two dogs were sensitized to cow's pigment and were subsequently perfused with cow's liver, kidney, and heart-muscle extracts. Neither dog showed any reaction. The protocol of one such dog, 17–32, is given.

Feb. 6. Sensitized by intraperitoneal injection of 7*cc* cow's uveal pigment emulsion.

Feb. 28. Perfusion started at 11.00 A.M. Head washed out as usual. Perfusion fluid 800*cc* normal dog's defibrinated blood, 400*cc* salt solution.

11.00 A.M. Pupils are 10*mm* in diameter. Fundi are negative.

11.15 A.M. Pupils are 10*mm*. Fundi are negative. 100*cc* of a mixture of cow's liver, kidney, and heart-muscle extract added to perfusion fluid.

11.30 A.M. Pupils are 10*mm* in diameter. Fundi negative.

11.45 A.M. Pupils are 10*mm* in diameter. Fundi negative.

12.00 M. Pupils are 9*mm* in diameter. Fundi negative.

12.15 P.M. Pupils are 9*mm* in diameter. Fundi negative.
12.30 P.M. Pupils are 10*mm* in diameter. Fundi negative.

These dogs were sensitized to the pigment alone, and protein other than pigment, from the same animal, could not produce intoxication. The pigment is not species specific in this ocular reaction.

DISCUSSION.

Von Szily was unable to demonstrate general anaphylactic reactions by sensitization and reinjection of heterologous uveal pigment in rabbits. In the light of our experience with pigment prepared by the method used by von Szily, it seems probable that his failure to obtain positive results may have been due to the effect of heat upon the pigment. The pigment preparations dried by heat and the preparations dried in a vacuum, but heated to 80° in the preparation, in our hands reacted with irregularity, and were moreover unsuitable for use in perfusion. It seems probable, however, that the effect of heat upon the pigment was to destroy, or at least alter, its antigenic properties. It may be that with us excessive heat was used, but the regularity of the reaction of the unheated pigment, compared to the large number of failures and irregular reactions obtained when the pigment was heated even to 80°, indicate strongly that heat destroys both the absorptive and antigenic properties of the pigment.

The establishment of the property of organ specificity and lack of species specificity for uveal pigment is of great interest in relationship to the chemical nature of the pigment. From the immunological standpoint, the pigment stands apart from other protein complexes of the body, and is analogous to similar pigment, irrespective of the species. A somewhat similar analogy is shown in its chemical composition. Samuely (11) sums this up as follows:

"In this group [melanins] are brought together black and brownish black bodies which occur in both vertebrate and non-vertebrate animals, under normal and pathological conditions. The correlation of the black pigments into a single group is not alone on account of the color characteristics, but is also justified on account of the highly probable general

conformity of their composition of cyclic complexes arising from the albumen molecule. These [pigments] arise partly out of colorless chromogens which are the same sort of aromatic and heterocyclic albumen derivatives, and which by their change into melanin . . . can draw in . . . accessory groups which contain sulphur and iron complexes, or even branched aliphatic chains."

In a previous paper, in relation to organ specificity, we have commented upon the "chemical affinity" to uveal tissue, irrespective of species, shown by animals sensitized to uveal tissue. With the establishment of the fact that the peculiar antigenic properties of uveal tissue are due to the pigment, it is of extreme interest that a probable cause for this can also be shown.

The ability of uveal pigment to act as antigen to the homologous animal is also paralleled by lens protein. Uhlenhuth has shown that lens protein can act as foreign protein to the homologous animal, and produce a specific hypersensitiveness. The property of organ specificity has also been demonstrated for testicle and placenta protein.

The relationship of these findings to the anaphylactic theory of sympathetic ophthalmia has already been emphasized. The anaphylactic theory assumes that uveal tissue absorbed from the exciting eye acts as an antigen and creates a hypersensitiveness, especially of the second eye, and anaphylactic intoxication of the second, manifested as a uveitis is brought about by the reaction of the antibodies formed in the cells, with the circulating antigen. Until Elschnig brought out his complement fixation work there was no reason to believe the theory a possibility for it assumed points not established scientifically. These points were ability of uveal tissue to act as antigen, organ specificity, and lack of species specificity. Elschnig claimed by means of complement fixation to have shown that uveal tissue possessed these properties. The work performed in this laboratory, with a different, and more fundamental, method, has yielded these same conclusions.

SUMMARY.

A pigment emulsion has been prepared from the uveal tract, which is suitable for use in perfusion. This pigment is a

nitrogen, hydrogen, carbon, oxygen, iron, sulphur, and phosphorus-containing body, and is either a protein or closely allied to proteins. Perfusion experiments proved that this pigment was the constituent of uveal tissue responsible for its peculiar antigenic properties. The pigment possesses antigenic properties, can act as antigen in the homologous animal, and is organ specific, and not species specific.

These antigenic properties of pigment are those required if the anaphylactic theory of sympathetic ophthalmia is a possibility. The findings reported here confirm Elschnig's findings, and give a definite scientific basis to the anaphylactic theory of sympathetic ophthalmia.

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GLIOMA RETINÆ ET ATROPHIA BULBI.[1]

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(*With three illustrations on Text-Plate VIII.*)

MORE than a century ago the neoplasm now known as glioma of the eyeball was called fungus hæmatodes. In 1809 Wardrop (1 A) wrote the first accurate and for many years the best clinical and most scientific description of the disease. After a study of seventeen cases he dwelt extensively on the metastatic and malignant character (1) of the growth and much about the glandular affections (1 B), and even advised and practiced early removal of the tumor. The value of his work was disregarded and but poorly appreciated till in 1853 Virchow and Von Graefe made exhaustive clinical and pathological researches relative to the disease; and the frequent references of Virchow to the pioneer work of Wardrop well attests the regard and appreciation which the master mind of pathology had for the early writer. Virchow held that glioma in its relation to brain structure is similar to elephantiasis in its relation to skin structure (2, pp. 151–157). The etymology of the term glioma is sufficiently suggestive, and at the time when Virchow announced his neuroglia theory, he also indicated the resemblance macroscopically of a glioma and the white substance of the brain; this analogy Wardrop had also observed and recorded (1, pp. 16–17).

The term glioma retinæ (2) as applied to tumors of the

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ILLUSTRATING DR. VERHOEFF'S PATHOLOGICAL REPORT ON DR. O'CONNOR'S CASE OF "GLIOMA RETINÆ ET ATROPHIA BULBI."

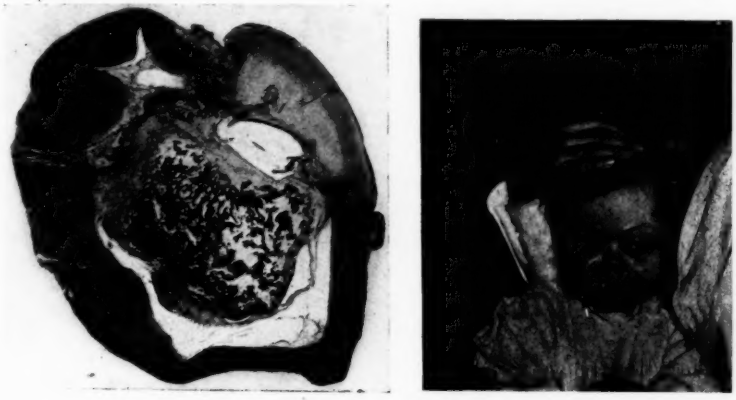

FIG. 1.—Section through center of globe showing vitreous chamber filled with connective tissue containing calcified masses of tumor cells.
Photo magni. x 5.

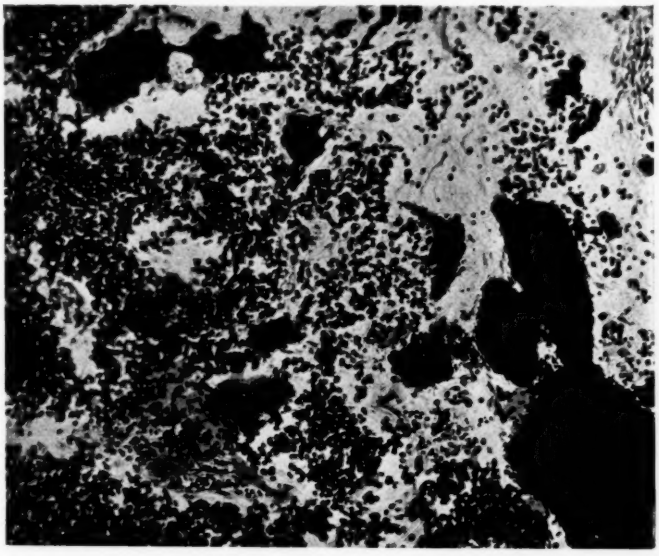

FIG. 2.—Showing tumor cells undergoing transformation into calcified masses.
Photo magni. x 150.

retina many years ago by the eminent Virchow, although a misnomer, has been retained in this instance on account of its general use, and as more comprehensive than the nomenclature of Flexner and Wintersteiner which seems rather restricted to tumors originating in a specific layer of the retina (3). A glance at the literature of gliomatous growths from 1870 to more recent times will show reliable reports from pathologists whose cases indicate manifold variations from those in which the retina was entirely obliterated (4, and 6 B, Case iii., and 1, p. 14) in the tumor mass to another in which the layer of rods and cones was demonstrated to be clearly free from participation in the pathological process (5).

Nearly half a century ago Herman Knapp (6 A) referred to the retina as "the most sensitive interpreter of pathological processes," and, he states that "there is no better site in the human body to trace and observe the development of a tumor than in the retina." He made a further plea for the intensive study of intraocular tumors on account of his conviction that they are essentially adapted for scientific observation and research as to the origin, progress, and result of malignant growths in general, as well as the theories and therapeutics pertaining to them (6).

The personal equation is well exemplified in the manner the laity regard the discovery of gliomatous growths. The man who disregards certain skin affections which are the obvious forerunners of malignancy, or the mother who neglects to report a growing nodule in her breast, will hasten to the oculist in great alarm with the child in whose eye the false gleam or reflex has been discovered shortly. The amaurotic cat's eye (7) is usually the first indication that hastens a parent to seek advice and relief for children afflicted with glioma retinæ.

But at times there is no such storm signal, or, if so, it is not properly observed; consequently, the symptoms become masked, the diagnosis obscure, and disaster to life is the result. The case reported here involves both the obscure disease in one eye and the obvious disease in the other.

The patient was seen by me several years ago and a pathological examination of the eye was made by Doctor Verhoeff. Recently while looking over the literature of glioma retinæ,

I concluded that the case related here presents some features which seem worthy of note.

A nine-months-old female infant was brought to the Eye Clinic of the Worcester City Hospital and a vague and unsatisfactory history of the eye trouble was obtained. The patient was a well nourished, healthy-appearing female child of whom the mother reported that a few days after birth the right eye became "bloodshot with a slight discharge." The child had rubbed the eye continuously and the mother attributed all subsequent trouble to the irritation produced by the rubbing. No history of ophthalmia neonatorum or other specific disease was elicited or later suspected. Through the courtesy of Doctor Harrower I am able to state that he had seen the case six months previously in his office, and then the affected eyeball was of normal size but the anterior chamber was filled with blood. During my examination the child continued to rub the diseased eye at intervals.

Examination.—The right eyeball is greatly shrunken; tension—2; inflammatory symptoms have apparently subsided; the sclera is dull and mottled; pupil is of medium size; iris is atrophic and adherent posteriorly, of dull green color in contrast to the light blue of the healthy eye; through the pupillary area a white object like a calcified lens is seen. *Diagnosis*: phthisis bulbi. Removal advised.

Left eye: horizontal nystagmus; pupil clear; no sign of inflammatory disturbance; eye follows objects moved before it; vision is apparently good. Four months later the shrunken eye seemed to be clearer and more quiescent than at the first visit. Seven months after I had first seen the child, the mother brought it to my office and said that the child had been rubbing the healthy eye continually; this habit had annoyed the parent so much that she was resigned to the removal of the shrunken eye, because she remembered that rubbing was the cause of all the trouble in the bad eye. The good eye had now become sore in a similar manner and she feared the same result with this one. The atrophic eyeball was practically unchanged from the last time I saw it; the other eyeball was reddened and quite sensitive to touch. No ophthalmoscopical examination could be made on account of the irritability of the child. I supposed that we were dealing with a case of sympathetic ophthalmia. The phthisical eye was shortly after removed at St. Vincent Hospital; the orbital tissues were inspected and to all appearances were healthy and not involved in any pathological change. After the operation the child collapsed and artificial respiration and stimulants were required for some

time. The enucleated eye was sent to Dr. Verhoeff for microscopic examination; he returned a diagnosis of glioma retinæ et atrophia bulbi. The remaining eye continued to show signs of irritation; a hypopyon developed, and two weeks later a white reflex appeared in the lower nasal quadrant of the eye, and in consultation with Dr. Harrower it was recommended to remove the eye for probable glioma,—a suggestion which was promptly rejected by the parents. The child was treated elsewhere for some time and eventually drifted into the clinic of Dr. Verhoeff at the Massachusetts Eye and Ear Hospital; he recognized the case and advised that the child be sent back to us for operation on the second eye. This advice was unheeded, and not till ten months after the first operation, and more than nine months after the diagnosis of glioma in the second eye had been established, did the case come under my observation and treatment again; the mother then returned with the child, pleading that "something be done before the eye popped out of the head."

The eyeball protruded almost outside the lids and rested on a mass of red and œdematous tissue that resembled beefsteak; the cornea was lusterless and collapsed, but a yellow gleam was visible through the pupil.

The child continued to rub this œdematous mass incessantly.

The facial veins on the affected side were dilated, the cervical and preauricular glands were enlarged; a small swelling like an erupting tooth was noted on the right upper jaw behind the first molar tooth; and, several days later, an area resembling a diphtheritic exudate appeared at this site, and extending about an inch long it involved the entire width of the gum.

Operation.—This consisted of removing the entire contents of the orbit; the cornea perforated and emitted a mass of fungus hæmatodes. The child bled through the nose profusely during the operation, and when the soft parts were removed, the lacrimal bone and nasal process of the superior maxilla were found to have been involved and the child breathed hissingly through the naso-orbital opening.

Following the operation a sero-purulent discharge appeared in the orbit and adjacent parts; the neoplasm started to grow from two distinct points in the orbit and enlarged rapidly; the metastatic area on the right gum grew till it pushed the teeth toward the middle of the mouth and developed a slough. At this time a reddish mass made its appearance in the orbit of the right eye. The patient died of sepsis and exhaustion nearly two months after the operation.

A meager necropsy was permitted and Dr. Baker reported the following: "The body is emaciated, face distorted; marked prominence on both sides of upper jaw due to growths which make nose appear flattened; necrotic black area size of fifty-cent piece over right upper angle of mouth; most of the teeth of upper right jaw are loosened or have dropped out; foul gangrenous mass over right half of hard palate. The skin of the face is œdematous over the growth which involves the entire superior maxillæ and renders them nearly twice their normal size; this includes mostly the region of the antra which are filled with a foul suppurating mass of tissue. The fleshy parts covering the superior maxillæ have been destroyed and spicules of bone like porcupine quills remain. The entire part exposed was saturated with pus so foul in odor that it drove the spectators from the room.

MACROSCOPIC EXAMINATION OF LEFT EYE (8).

The eyeball is elongated and oval-shaped; it measures:

Antero-posteriorly	33*mm*	Adult normal eye is	23.9*mm*
Vertical diameter,	23*mm*	" " " "	21 *mm*
Transverse diameter	21*mm*	" " " "	23 *mm*

The average weight of the human eyeball is 7 grammes; this eyeball weighs over 31 grammes. The sclera of this eye is 1*mm* thick, and attached to the naso-inferior part of it lies an area of raw red tissue which measures 7*mm* wide and extends along the entire eyeball: this part lay on the lacrimal bone and involved it.

Unfortunately this eye was lost by a physician who undertook to photograph it, so no microscopical specimen was made of it; the picture here shows this eye a day or two before it was removed.

PATHOLOGICAL EXAMINATION OF RIGHT EYE.[1]

By Dr. F. H. Verhoeff.

(2–1607) The globe was fixed in 10% formalin and decalcified in 8% nitric acid. It measures 11*mm* anteroposteriorly, 10.5*mm* horizontally, and is greatly distorted, presenting the external appearances ordinarily produced by phthisis bulbi (Fig. 1). There are no evidences of per-

[1] From the Pathological Laboratory of the Massachusetts Charitable Eye and Ear Infirmary.

foration. The cornea is thickened to *2mm*, while the sclera is wrinkled and in places is *2.5mm* thick. The anterior chamber is obliterated. The lens is shrunken to *2mm* × *3mm* in size. The vitreous chamber is completely filled with tissue, containing numerous small calcified areas, which in places has retracted from the sclera a distance of *1mm*. The choroid, retina, and iris can not be recognized macroscopically.

Microscopic Examination.—The epithelium of the cornea, together with Bowman's membrane, shows numerous fine folds. Descemet's membrane is intact, but likewise shows numerous fine folds and in addition many large ones. The thickening of the cornea is not due to cicatrization of the stroma, but evidently to contraction of a layer of fibrous tissue which separates the cornea and lens. This tissue is not pigmented and bears no resemblance to iris tissue. On one side no traces of the iris remain and the fibrous membrane continues into the highly atrophied and almost unrecognizable remains of the ciliary body. On the other side it continues into a small amount of disorganized pigmented tissue. This is no doubt remains of the iris, since it is connected with the ciliary body which on this side is not so greatly altered. From the ciliary body on both sides arises a dense fibrous membrane which is applied to the posterior surface of the lens and joins the fibrous membrane in front of the lens so that the latter is completely enclosed in fibrous tissue. The lens capsule is greatly wrinkled and folded, but is intact except at one place. Beneath the anterior capsule there is a thick almost uniform layer of lime salts which extends a little beyond the equator. The capsular epithelium has disappeared. The lens substance is replaced by delicate avascular connective tissue which is connected with the cyclitic membrane through a break in the capsule at the equator of the lens.

The choroid has been converted into fibrous tissue and is recognizable only by the persistence of the pigment epithelium. The latter is seldom in the form of a single layer of cells but shows marked proliferative changes. The choroid shows no calcification or ossification. The optic nerve is greatly atrophied and loses itself in the tissue within the globe. No traces of the retina can be found. The small vitreous chamber which remains is filled with delicate œdematous connective tissue containing numerous sharply defined granular masses. These stain intensely in hæmatoxylin and otherwise present the usual picture produced by deposits of lime salts. The connective tissue is continuous with the choroid through breaks in the pigment epithelium and is almost free from blood-vessels. Within this tissue,

at the posterior part of the eye, is a mass of cells occupying an irregular area about *2mm* in diameter. The cells show a distinct tendency to group themselves into irregular alveoli. At one point they have invaded the choroid and formed a small nodule within it. On close examination the cells are seen to be similar in appearance to those most commonly found in a so-called glioma retinæ. Their cytoplasm is scanty and many of them show small processes at one or both ends. The average diameter of a cell is about twice that of a lymphocyte. Their nuclei are generally slightly ovoid or pear-shaped. No rosettes are present and there is no tendency for the cells to take on a perivascular arrangement, probably owing to the scarcity of blood-vessels. In places sharply defined groups of tumor cells are found nudergoing calcification, so that it is evident that the granular masses everywhere consist of calcified tumor cells (Fig. 2). Such calcified masses are being formed also in the small nodule of tumor cells within the choroid, which evidently is of comparatively recent origin. The eye is practically free from inflammatory reaction, showing only an insignificant infiltration with lymphoid cells in places.

Pathological Diagnosis: Glioma retinæ et atrophia bulbi.[1]

REMARKS.

That this is a case of glioma retinæ in a shrunken globe is beyond doubt. The question arises, however, whether the phthisis bulbi or the glioma was the primary condition. Against the former possibility is complete disappearance of the retina, the character of the tissue filling the globe, and the history of the case. Assuming therefore that the glioma was primary, how may the condition be explained? There is no difficulty in accounting for the marked inflammatory reaction necessary to explain the cicatrization of the tissues of the eye, for this is frequently observed not only in cases of glioma retinæ but also in cases of choroidal sarcoma in which the tumor has undergone necrosis. At first glance it would seem that the calcified masses represented cell masses of the original tumor that had become necrotic, but more careful consideration leads me to believe that this is not the correct explanation. For I have never seen such appearances within the large

[1] The term atrophia bulbi is used here because it has generally been employed in such cases by other authors, but as a matter of fact the condition more closely resembles phthisis bulbi.

necrotic areas of a glioma retinæ, while, on the other hand, I have seen in the œdematous connective tissue at the periphery of a necrotic area the identical picture presented in the present case. It would seem, therefore, that in this case the tumor, after destroying the retina, became almost completely necrotic, leaving only here and there living tumor cells. As the necrosis went on, the necrotic areas became replaced by connective tissue, and in this tissue the tumor cells again grew. The cell masses thus formed then underwent necrosis and calcification, probably from the lack of sufficient blood supply.

HEREDITARY, PRENATAL, AND CONGENITAL ASPECTS.

Von Graefe (9) in his masterly style has given such an adequate and comprehensive description of the clinical course of gliomatous growths that even in our day repetition too often replaces originality and here it seems superfluous.

A few points of peculiar interest from other authors may be pertinent as to the hereditary, prenatal, and congenital aspects of glioma.

While heredity seems hardly probable, still some records show the presence of the disease in all the children of certain families (10).

One report indicates that eight children of the same parents were afflicted with, and died in succession from, glioma retinæ (11).

Newton states that ten of a family of sixteen members had glioma retinæ and all but one of the patients died before the age of three (12).

Another author describes two cases of retinal glioma among four children of the same mother (13). Von Graefe (9) reported two instances of the neoplasm in members of the same family; the mother of the victims had lost several brothers or sisters who had died in infancy from "cancer of the eye."

Lerche contributed the history of four out of seven brothers and sisters who had been afflicted with and died from glioma retinæ (14).

Mr. Travers described and presented engravings of a child suffering from this disease whose eyeball was as large as a

walnut at birth and at eight months was the size of a man's fist (15).

Fuchs relates the case of a child of four years with glioma retinæ which had existed for a year; death resulted one year after evisceration of the orbit. Some time later the mother brought her second child to the clinic and he too was similarly afflicted and died; subsequently she returned with her last baby of several months. The horrible disease was absent in this case, but the infant had a coloboma of the iris and choroid (16).

The possible prenatal origin of the above cases calls to mind the statement of Treacher Collins that a microscopical specimen of a fœtal retina at three months with layers undifferentiated is such that if shown to a pathologist it would be pronounced by him to be glioma retinæ (17).

A case which Parsons classes as dubious was reported of a child whose eye was removed for glioma, living to adult age and having children afflicted with glioma (18).

The family history of our case disclosed no record of neoplasm, ocular or somatic, on the side of either parent as far back as they could recall; other children in this family are all alive and healthy.

METASTASES.

That a metastasis does not occur early in many cases may be inferred from the case of Geissler, who in 1871 described a gliomatous growth in an eyeball which did not have time to extend to any part of the cranium before the child died of exhaustion (19).

Von Ammon depicted a child of one and a half years who had a metastatic growth on both surfaces of the frontal bone from a bilateral glioma (20).

Chisolm (21) reported a case some years ago wherein there were numerous metastases, but the new growth remained practically stationary for months while the patient suffered severely from cholera infantum; the eyeball remained unchanged despite œdema and bulging of the orbital contents. A swelling appeared on the lower left jaw and grew so more rapidly than all the other metastases and enlarged in length

and breadth to such an extent that the chin was pressed to the right side. Death occurred one year after the first visit to the clinic and six months after the first appearance of metastasis.

De Gama Pinto (22) mentions a case of glioma with metastatic involvement of the parotid region and extending from the auricle to the angle of the jaw; the growth was 3½*cm* wide and 5*cm* high.

An interesting illustration of metastasis is given by Battman (23) of a bilateral glioma retinæ wherein tumors appeared on the upper jaw, then in the cheeks and on the forehead; metastatic deposits were also found within the cranium, on the sella turcica, and other parts.

Secondary deposits of a gliomatous nature were found in the periosteum of the facial and cranial bones by Watson (24).

So far as we were able to ascertain, the metastases of the case described by us were limited to the head; the growth and sloughing of the right gum were on the side opposite to the second eye affected, *i.e.* on the side of the atrophied eyeball, whose orbital contents had shown no pathological activity from the date of enucleation, ten months previously, till the second operation, when an inspection revealed a red area about 10*mm* in diameter over the site of the nerve stump. The necropsy report indicated the antrum on each side was filled with a "foul suppurating mass of tissue."

The ability to produce atrophy in an eye afflicted with glioma, as deduced from the case histories and the treatment advocated by ophthalmologists of a hundred years ago, was evidently an article of faith among them. Sichel (25) states that atrophy of an eye with true encephaloid can be brought about by antiphlogistic, alterative, and derivative treatment. Calomel, blue mass, inunctions of neapolitan ointment, black oxide of copper ointment, chloride of barium, antimony, and the iodides furnished the greater part of his armamentarium for attacking the disease; and, he declares: "Many times I have arrested the progress of the encephaloid by the employment of this treatment and obtained atrophy with no single occurrence of the disease." Herman Knapp (6 C) mentions the attempts of oculists in the middle of the last century to cause atrophy of the "pseudoplasma" with appropriate medical treatment. Virchow (26) did not regard the

spontaneous cure of glioma as impossible. He held that the temporary shrinkage of an eye in some forms of glioma is produced by a high-grade metamorphosis and absorption of the fluid elements. Lagrange (27) in his review of twenty-one cases shows that retrogressive metamorphosis is not a myth.

Von Ammon (28) showed in several figures the successive stages of probable fungus hæmatodes in which the vascularity abated and the enlargement subsided and the globe was in a state of complete atrophy at the end of six years. Schweigger (29) states that frequently in the course of glioma changes occur which end in atrophy of the eyeball, but later he qualifies the statement by saying that the course of the disease is not generally checked by the intercurrent shrinking.

Stellwag von Carion (30) refers to the glaucomatous epoch of the growth as: "Having reached this stage, or sooner, sometimes, the growth of the tumor ceases, and the process is arrested. Not infrequently it recedes; the formerly tense globe becomes softer and atrophied."

Beyond all doubt, glioma is capable of retrogressive metamorphosis which manifests itself through fatty degeneration and the globe remains for life in the state above described. However, he suggests that in some cases sooner or later the growth returns to the detriment of the patient.

Hirschberg and Happe (31) described a case many years ago with which the case related by us has much in common. A child twenty weeks old was brought to their clinic, and on examination it was found that the right eye was soft and shrunken; the anterior chamber was filled with blood. A white reflex had been noticed in the eye some few days subsequent to birth and the atrophy of the globe was subsequent to perforation of the cornea from the growth within. Enucleation was done when the child was seven and a half months old. One year later the patient was returned to the clinic with the same symptoms in the left eye and this went on to destruction. There was no recurrence in the right orbit one year after enucleation.

Thirty-four years later, Hirschberg (32) reviewed some seventeen cases, of which ten at the time of operation had not extended beyond the retina, and in none of these had a relapse occurred during a period of observation from five to twelve

years subsequent to operation. The enucleation in all ten cases had been done within ten weeks from the first detection of the white reflex.

In the case cited above enucleation was not done till the child was thirty weeks old, and no recurrence was evident one year after the eye was removed. The effect of corneal perforation and partial evacuation of the ocular contents in these cases evidently has some inhibitive effect on the spread of the growth in the eye itself and also on the subsequent metastases extra-ocular. However, in the case related by us no perforation of the cornea or other part of the eye occurred at any time.

A satisfactory microscopical examination of the atrophic eyes was made in seven of the fifteen cases collated by Parsons (33), two others were doubtful glioma, and in the remaining six cases no microscopical examination was made. The macroscopic report of the non-microscopic cases lends little value to their credit, as follows: 1st "White tumor of retina; optic nerve normal"; 2d "Filled with growth; extra-ocular growths; optic nerve enlarged"; 3d "Large growth in the orbit"; 4th "Undoubtedly glioma"; no macroscopic or microscopic reports are given for the other two cases.

In ten of the fifteen cases the glioma was bilateral; in three there was perforation and shrinking. Consequently there was reason for Parsons to close his paragraph on atrophia bulbi with the statement that, "I am still of the opinion that the gliomatous nature of the cells in these shrunken eyes is not placed absolutely beyond cavil." Fortunately in our case Dr. Verhoeff made the microscopical examination and later saw the patient in the clinic at the Massachusetts Charitable Eye and Ear Infirmary.

As one may infer from the above, the literature and textbooks are replete with references to atrophy of the eyeball in glioma, as the final result or as marking the temporary cessation of progress with the neoplasm. One may well regard many of these cases as pseudogliomata or simply as an episode in the course of a true glioma, preparatory to further advance.

DIFFERENTIAL DIAGNOSIS.

The differential diagnosis of glioma may offer a few items of interest and at times afford opportunity for clever discrimination between facts and fancies. De Schweinitz (34) reported a case of retinal detachment with extensive dropsical degeneration of the rods and cones which exactly simulated glioma retinæ. A corneal cicatrix or staphyloma may obstruct the pupillary space, or in the glaucomatous stage of glioma, a hyphæma or cataract may obscure or prevent ophthalmoscopic examination of the eye affected.

A cysticercus, though seldom seen in children and rare in this country, is another possibility.

A hemorrhage into the vitreous may become organized and be confused with glioma, or a metastatic uveitis from meningitis or other infectious diseases may cloud the diagnosis (35).

Persistent vascularity of the lens capsule, or a hyaloid artery with posterior cataract has been found in eyes enucleated for glioma retinæ (36). A report from Moorfields Hospital from 1888 to 1893 indicated that seven of twenty-four eyes enucleated for glioma were found to be pseudo-growths (37). A detached retina bulging forward may occlude a tumor of this kind and render diagnosis difficult, according to Schmidt-Rimpler (38), who then calls attention to the larger blood-vessels and irregular course of them in glioma. Jung (39) did an iridectomy on a child of four years after a diagnosis of tubercular iridochoroiditis had been made; this, perhaps, was prejudiced by the fact that the mother was phthisical. Amaurotic cat's eye, hypopyon, and nodules developed later and a crypto-glioma was apparent.

The atrophic eye of our case, when seen by me on several occasions, seemed like an ordinary shrunken globe, subsequent to severe inflammatory process, and removal was suggested only because of the probable danger to the good eye from sympathetic involvement.

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SARCOID OF THE EYELID.

By Dr. GEORGE DERBY, Boston.

PATHOLOGICAL EXAMINATION.

By Dr. F. H. VERHOEFF, Boston.

(*With three illustrations on Text-Plate IX.*)

IT seems worth while to put on record the following case, as I have been unable to find in ophthalmic literature any reference to sarcoid, and in a fairly extensive search in the literature of this disease I have not happened upon a case where involvement of the ocular structures was noted.

N. H. female, aged 25 (?), Irish by birth, was seen at the Massachusetts Charitable Eye and Ear Infirmary in December, 1912. Three months previously she had noticed a small growth in the outer half of the right upper lid, which was painless, but had been gradually increasing in size. As a small child she had received a slight injury, represented by a small scar in the skin at the outer canthus. Furthermore, there was a rather indefinite history of an inflammation in childhood, and each cornea showed a faint nebula near the center. Vision with correction $\frac{6}{12}$ in each eye, otherwise the eyes were normal.

The growth of which the patient complained was situated in the outer third of the lid, and ran upward and inward under the orbital margin. It was 2.5 to 3*cm* in length and approximately 4 to 6*mm* in breadth. It was flattened and slightly lobulated in appearance, as though made up of nodules which had coalesced. It was situated deeply in the skin, and formed a very distinct and hard thickening, almost cartilaginous to the touch. In color, it was a deep yellowish-red.

The patient was taken into the hospital, and the growth excised by Dr. E. E. Jack. Three months later, she returned to the hospital with a very distinct recurrence extending

ILLUSTRATING DR. VERHOEFF'S PATHOLOGICAL REPORT ON DR. DERBY'S CASE OF "SARCOID OF THE EYELID."

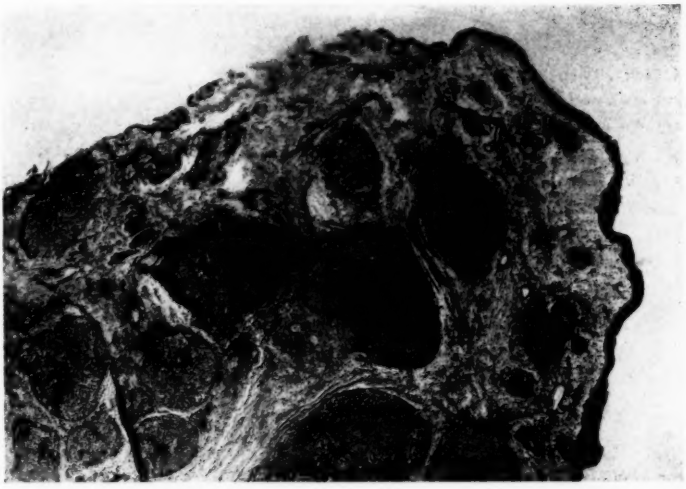

FIG. 1.—Showing tubercles at periphery of nodule in skin of eyelid.
Photo x 22.

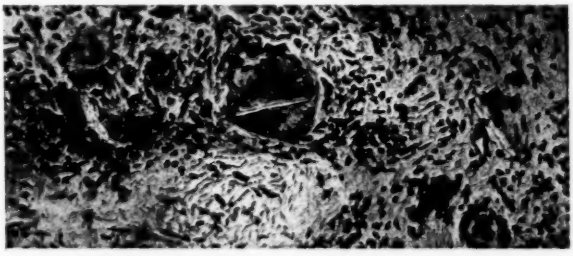

FIG. 2.—Showing three giant cells each containing a crystal.
Photo x 180

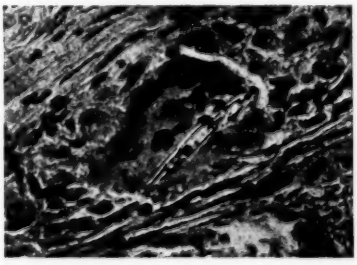

FIG. 3.—Giant cell containing a crystal.
Photo x 375

over the same area as before, and in addition she showed a discrete nodule of the same type situated on the right cheek. She then came under my charge, and was referred to the Dermatological Department of the Massachusetts General Hospital, where no diagnosis could be made. She was again admitted to the Infirmary, and both areas excised, care being taken to remove all tissue that appeared abnormal. Healing was prompt, and the patient was discharged two days later in good condition. There has been no return of the disease. The excised tissue was examined by Dr. F. H. Verhoeff, who reported as follows:

(3–2282) The specimens submitted for examination consist of two pieces of tissue removed at different times from the eyelid, and a nodule removed from the cheek. One portion was fixed in Zenker's fluid, another in 10% formalin, and the other in absolute alcohol.

The cut surface of the hardened tissue showed a distinct alveolar structure suggesting that of a lacrimal gland. Celloidin imbedding was employed, and the sections stained in hæmatoxylin and eosin and a variety of other stains.

In cutting the sections, it was observed that the edge of the microtome knife soon became injured owing to the presence of gritty particles in the tissue. Cover slip preparations were also made of scrapings from the surface of the hardened tissue.

On microscopic examination all of the sections show the same appearances. There is seen a conglomeration of tubercles, partly or wholly separated from each other by septa of connective tissue. All of the tubercles seem to be situated in distended lymph spaces, the involvement extending through the entire thickness of the skin into the underlying fat tissue and muscle. The tubercles are always separated from the surface epithelium by a layer of connective tissue. Under the low power the general configuration closely simulates the appearance presented by an epithelioma of the skin which is invading the lymph spaces (Fig. 1). The tubercles vary greatly in size, the largest occurring in the superficial lymphatics, and the smallest in the deepest layers and at the periphery, where the process is evidently extending. The tubercles are made up of typical epithelioid (endothelial) cells, which have gone on to the abundant formation of giant cells, many of the latter being of the Langhans type. The larger the tubercles, the larger and more numerous the giant cells. Blood-vessels are only occasionally seen within the tubercles. The tubercles show no infiltration with pus cells, but there is slight infiltration with lymphoid cells, generally at their peripheries. There is

no necrosis of any kind to be seen. The stroma between the tubercles is notably free from inflammatory reaction.

Sections stained for tubercle bacilli fail to show any organisms present.

On close examination it is found that the tubercles contain crystals and amorphous precipitate. The latter is greenish yellow in color, while the crystals are almost colorless. Usually the crystals have a greater or less number of the amorphous granules clinging to them, which renders them more readily visible. In fact, they are very difficult to see when they are free from these granules. In shape a typical crystal seems to be in the form of a needle pointed at one end only, but many malformed crystals are seen. The crystals and amorphous precipitates occur almost exclusively in giant cells (Figs. 2 and 3). In size the crystals vary from about 40μ to 70μ in length and average about 4μ in thickness. In the smaller giant cells they are apt to occur singly, but in the larger giant cells several may be clustered together. They are much more abundant in some places than others, but careful examination shows that they are present in all the tubercles.

Diagnosis.—Sarcoid of the skin.

Remarks.—The sharp definition of the tubercles from the surrounding tissues and the lack of inflammatory reaction in the latter suggest strongly that they are due to the presence of some sort of foreign bodies. The crystals and amorphous precipitate found within the giant cells are sufficiently abundant to explain the tubercles. That these precipitates have not previously been seen may be due to the fact that they have been less conspicuous in other cases. The possibility suggests itself that they may have been formed as a result of an inflammatory process. Against this, however, is the fact that they do not occur in tuberculous and other processes where the formation of endothelial cells is equally abundant. That they are not artefact is indicated by the fact that they occur in all three specimens, each of which was fixed by a different method, and also by the fact that they had evidently caused the giant-cell formation. It is possible, therefore, that the process may have been due to some obscure metabolic disturbance leading to the localized deposition of crystals and precipitates in the tissues of the skin. This would, in a general way, be analogous to the formation of tophi in gout, although the inflammatory process does not otherwise resemble that of gout.

Although no diagnosis could be made at the time, the case was kept in mind, and finally Prof. S. B. Wolbach, of the

Harvard Medical School, after viewing the sections, suggested that the condition was that described by Boeck.

The term sarcoid was originally used by Kaposi to describe a number of different pathological conditions of the skin, including lymphodermia, mycosis fungoides, and even true sarcoma. In its present sense, it was first employed by Prof. Boeck, of Christiania, in 1899.

The disease may begin either as a diffuse œdematous reddening of the skin, which later subsides, leaving one or several circumscribed and infiltrated areas, or it may appear in the form of one or more small nodules sharply circumscribed and situated deep in the skin and raising the epidermis. These nodules may increase in size and finally join, forming large, flattened, irregularly outlined patches. The lesions are prone to appear in crops, and may last months or years. As retrogression sets in, they may become umbilicated in appearance, and finally disappear, leaving pigmented spots or sharply defined white cicatrices. They never break down and ulcerate, but may show a slight scaling. In color at first red, they later assume a violaceous tinge with marked teliangiectases, and then become a yellowish-brown. Under glass pressure, in all forms of the disease miliary foci may be always distinguished, situated deeply in the skin and forming the most characteristic feature of the disease. The lesions may vary in number from a single one to many thousands. They occur by election on the face, back of shoulders, and extensor surfaces of the arms, but they may occur on any part of the surface of the body, and Boeck reports a case in which the mucous membrane of the nose was involved. Involvement of the lymph glands may or may not take place. The disease usually occurs between the ages of thirteen and forty-five, but has been reported in the earlier and later periods of life. Women seem to be more frequently affected than men.

Joseph reports a case in which there was general involvement of the inner organs and death took place, but in all probability it was a carcinomatosis of the skin.

In 1915, Kunitzky reported a case showing the typical sarcoid lesions in the skin and under the microscope, showing also marked cardiac, renal, and gastro-intestinal symptoms.

The lungs showed opacity to the X-ray, there were dyspnœa and rapid heart on exertion, the liver and spleen were enlarged, and there was albumen in the urine with hyaline and granular casts. The subcutaneous tuberculin test and the Wassermann test were negative, and there was no fever. As a result, the writers believe the condition to be wholly due to sarcoid.

In a footnote Kunitzky refers to an investigation in progress at the time of writing on seven other cases of this disease, all of which showed lung symptoms; four showed swelling of the spleen, three showed involvement of the mucous membranes (nose, larynx, pharynx, epiglottis). All gave negative results to large subcutaneous tuberculin tests.

Other writers have reported involvement of the inner organs. Terebinsky reported enlargement of the spleen in a somewhat doubtful case; Bering, involvement of submaxillary gland; Bloch, involvement of the iris; Boeck himself observed bronchitis and catarrh of the lungs; and general glandular involvement has also been reported.

The histological picture is as characteristic as is the clinical appearance. It shows circumscribed, deep-seated nodules throughout the whole depth of the corium separated from each other by connective-tissue septa. These nodules apparently have their origin in the perivascular lymph spaces, and are composed centrally of epithelioid cells with faint staining nuclei. In the periphery are a few lymphocytes and plasma cells. Giant cells are occasionally seen, and dilated capillaries are easily to be distinguished. The absence of caseation is characteristic. It remains for future examinations to show whether the crystals found in the case reported here are a constant feature of the disease.

Types.—Many of those who have observed these cases, including Boeck himself, have remarked on the various features of the disease which resembled tuberculosis. A number of cases have even been reported where the tuberculin reaction was positive, and others where undoubted tuberculosis was obtained in inoculated animals.

Darier, in 1910, published an extensive study of sarcoid, and divided the disease into four types:

1. Multiple benign sarcoid of Boeck, which again may be

divided into three forms: the large nodular, small nodular, and the diffuse infiltrated form.

2. Subcutaneous sarcoid of Darier-Roussy (subcutaneous tuberculide).

3. Erythema induratum-like sarcoid of the limbs.

4. Spiegler-Fendt type. Sarcoid with round cells, non-tubercular, and resembling neoplastic lymphoderma.

The multiplication of the cases reported and the closer study of the disease lead to the belief that the designation sarcoid should in general be confined to the type of disease originally described by Boeck, Type 1.

Type 2, of Darier-Roussy, is exceedingly rare. The lesions are situated in the subcutaneous tissue and differ markedly from Boeck's type in appearance and size. Until further evidence is produced, it can hardly be classed as sarcoid.

Type 3 resembles closely erythema induratum. The lesions occasionally ulcerate, the tuberculin reaction is sometimes positive, tuberculosis has been produced by inoculation, and Philippson has demonstrated tubercle bacilli in the specimens.

Type 4, of Spiegler-Fendt, although presenting clinically many of the features of Type 1, shows under the microscope collections of round cells, while epithelioid and giant cells are absent or rare. It seems probable that this type belongs to the neoplastic lymphoderma, but the resemblance to sarcoid is greater than is the case with Types 2 and 3.

The etiology of the true sarcoid of Boeck is still in the dark. While some writers regard it as a tuberculide, the weight of present opinion is on the other side. While sarcoid has till recently been regarded as a disease of the skin primarily, evidence is beginning to be brought forward to show that the disease at first is a general one and the cutaneous involvement is secondary. Kunitzky and Bittorf bring forward strong proof in favor of this assumption. It is therefore desirable that future cases should be carefully examined with reference especially to the condition of the mucous membranes, lungs, spleen, and liver.

For the diagnosis of sarcoid the microscopic examination is indispensable. The clinical appearances are also characteristic, but the following diseases should be taken into account:

syphilis, leprosy, leukæmia cutis, lupus erythematosis nodularis, erythema induratum, lupus pernio.

The prognosis for life is good. The duration of this disease entails weeks, months, or even years, and recurrences are not infrequent.

Boeck was the first to describe the almost specific action of arsenic upon these lesions, and other writers have been able to confirm his observations. X-ray has also been of service, and Darier recommends injections of calomel.

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THE PRESSURE SIGNS OF CERTAIN INTRACRANIAL CONDITIONS OBSERVABLE IN THE FUNDUS OF THE EYE.[1]

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THE fundus of the eye, and particularly the retina, being an offshoot of the brain is most intimately connected with the brain and the intradural cavity so that any lesion within the intracranial cavity which increases its normal content would naturally tend to be shown in the fundus of the eye, especially about the optic nerve head; that is, unless the normally free communication within the vaginal sheath between the intradural cavity and the optic papilla is obstructed by local disease, adhesions, etc., we should expect the signs of increased intracranial pressure to be observable in the fundus of the eye, particularly about the optic disk; again, an increased intracranial pressure sufficient to retard and even prevent the normal return flow of blood in the retinal veins would also tend to cause a dilatation of these retinal veins and the usual condition resulting from their dilatation and congestion.

The effect of an increase of intracranial pressure upon the fundus of the eye can be very easily demonstrated in its various stages by the experimental production of an internal hydrocephalus in dogs. During the past eighteen months, the condition of hydrocephalus was successfully produced in nine puppies of the age of ten days to two weeks; by means of a

[1] Read before the N. Y. Academy of Medicine, Ophthalmology Section, on March 19, 1917.

suboccipital exposure, a small gelatine capsule filled with cotton is inserted into the aqueduct of Sylvius so that the cerebrospinal fluid could not escape from the third and lateral ventricles; a resulting hydrocephalus with dilatation of the ventricles and therefore an increased intracranial pressure occurred so that it was possible with the ophthalmoscope to observe the changes in the fundus of the eye due to this increase of intracranial pressure. Within five to six hours after the insertion of the cotton plug into the aqueduct of Sylvius, in each one of the puppies the retinal veins gradually became dilated; apparently the veins over the nasal half enlarged earlier and possibly more than the veins over the temporal half of the retinal fundus; this congestion and dilatation of the retinal veins was the first sign indicative of an increase in the intracranial pressure. Within two to four hours later, the nasal margin of the optic disk would become blurred, then the temporal margin, then the nasal half and finally the temporal half of the disks would become obscured, so that within a period of twelve to twenty-eight hours following the production of a definite increase of intracranial pressure, the details of the optic disks could no longer be observed; in four dogs the œdema of the optic disks was so great within this period of time that a measurable swelling could be observed with the ophthalmoscope and thus the condition of "choked disks" could be applied; in the other five dogs, the measurable papillœdema occurred from twelve to twenty hours later. In one dog this condition of internal hydrocephalus with high intracranial pressure was not relieved by a subsequent drainage operation, and it is interesting to note that definite signs of a secondary optic atrophy began to appear nine weeks later; the remaining eight dogs were all drained through a subtemporal operation with six linen strands being inserted into the ventricle in order to relieve the condition of internal hydrocephalus; in all but three of the dogs, the increased intracranial pressure was relieved and it was most interesting to note that the subsidence of the choked disks was in just the reverse order of their occurrence, that is, the measurable swelling of the papillœdema first disappeared, then the blurring of the temporal half and then of the nasal half of the optic disk; then the temporal margin and later the

nasal margin appeared—though slightly blurred in all of the puppies; that is, the persistent dilatation of the retinal veins and some blurring of the nasal margin of the optic disks indicated that the intracranial pressure had not been entirely relieved by the operation. In the remaining three puppies the optic disks remained entirely obscured—only the measurable swelling disappeared, so that in these dogs the operation of drainage to relieve the condition of internal hydrocephalus was not successful.[1]

This experimental work has been most instructive to me regarding the mechanical factor in the production of papillœdema and the condition known as "choked disks." There exists at present much confusion in the terminology of retinal conditions especially in their relation to intracranial pressure; the terms papillitis and papillœdema have been used interchangeably, and "choked disk" most freely and carelessly. Naturally, the terms papillitis, retinitis, and optic neuritis imply a condition of inflammation of the nerve head and the retina. Inflammation (in its modern conception) is due to toxic and infective causes alone, so that in the condition of nephritis, diabetes, and the various forms of meningitis, the term papillitis would indicate a retinal change due to some toxic or infective cause, whereas the blurring and œdema of the details of the optic disk due to an increase of intracranial pressure in purely mechanical conditions, such as an intracranial tumor mass and hemorrhage, could be termed as a papillœdema; and if a measurable œdema, then a swelling of the disk up to the stage of "choked disk." In order that a choked disk occur it must always be preceded by a series of blurring of the details of the optic disk, so that these early œdematous blurrings of the optic disks have been termed the mild or early stages of papillœdema or even a choking of the disks. If it is believed that intracranial pressure alone does cause in the fundus of the eye an inflammation (using the term inflammation in its modern sense as being due to a toxic or infective cause), then the term papillitis is perfectly proper although the etiological factor in its production is pressure, but I do feel that the modern methods of examination of the cerebrospinal fluid, and especially its cell count, whereby any

[1] A detailed report of this experimental work will be published later.

inflammation and infectious condition within the cerebrospinal canal and also its projecting connections, such as the optic vaginal sheath, can easily be demonstrated,—that these methods should disclose the infective character of the cerebrospinal fluid if that is a cause of the so-called papillitis in the purely pressure cases. It has been frequently demonstrated in the body tissues and elsewhere that pressure can and does cause congestion and œdema, but not inflammation (in its modern sense), unless infection is present; pressure upon tissue renders that tissue more susceptible to inflammation by infection,—a predisposing cause,—but pressure in itself does not mean an inflammation. I believe it is rare for retinal hemorrhages to occur in cases of choked disks due to even extreme intracranial pressure alone, whereas in conditions of neuroretinitis, optic neuritis, and papillitis due to toxic causes such as diabetes and nephritis, these hemorrhages occur very frequently. However, if we must take it for granted that the retinal and optic-disk changes in conditions of intracranial pressure are due to both pressure and toxic factors,—possibly the pressure producing the toxic appearance of inflammation,—then the condition of blurring and œdema of the details of the optic disk would precede the stage of papillitis, and if the papillœdema became measurable to two or more diopters then the term "choked disk" should be applied. In discussing the pressure signs observable in the fundus in the following intracranial conditions, I shall use the terms blurring and œdema of the details of the optic disks to indicate the earlier stages of increased intracranial pressure, and its later stages of measurable swelling of the optic disk by papillœdema, and the "choked disks" in the conditions of extreme intracranial pressure where the papillœdema is greater than two or three diopters. These signs of increased intracranial pressure as exhibited upon the fundus of the eye have been checked up and confirmed by a measurement of the pressure of the cerebrospinal fluid at lumbar puncture by a spinal mercurial manometer, so that when there is observed a blurring or œdema of the margin on the nasal half of the optic disk in a patient following a head injury, or in a patient in whom a brain tumor or brain abscess is feared, or in a child following a difficult labor and convulsions occur so that an intracranial hemor-

rhage is suspected, then it is very important to record accurately the pressure of the cerebrospinal fluid at lumbar puncture; if the pressure of the cerebrospinal fluid too is shown to be increased and thus the ophthalmoscopic findings are confirmed, we can then reach a more accurate diagnosis of the intracranial condition. Naturally, in normal fundi blurring and mild obscuration of the details of the optic disk occur and are considered as being within physiological limits; especially is this true in myopia, but if this obscuration of the details of the optic disks is observed and then the measurement of the cerebrospinal fluid is performed at lumbar puncture by means of a spinal mercurial manometer (the most accurate method now known to record the pressure of the cerebrospinal fluid) we are thus enabled to exclude those cases of so-called normal blurring of the optic disks.

There are certain intracranial conditions that frequently produce definite pressure signs observable in the fundus of the eye. I should like to speak briefly regarding these pressure signs of the following intracranial conditions and in this order: brain tumor, brain abscess, intracranial hemorrhage and œdema following fractures of the skull, selected cases of cerebral spastic paralysis due to an intracranial hemorrhage at birth, and lastly the condition of hydrocephalus.

I. We all know the condition of choked disk and, if not relieved, its subsequent secondary optic atrophy in patients having the signs of intracranial tumor. Naturally for a choked disk to occur, there must be high intracranial pressure, and I believe it is rare in cases of brain tumor for a choked disk to result unless the tumor has become of very large size, or it causes a blockage of the ventricles, and thus produces an internal hydrocephalus, such as the posterior mid-brain tumors and the subtentorial tumors and cysts. It is comparatively easy to make the diagnosis of brain tumor at this stage of papillœdema, and I feel that if these patients had been examined ophthalmoscopically early, then the more mild pressure signs observable in the fundus resulting from the smaller tumor mass would have been ascertained and the patient thus spared an impaired vision, if not blindness itself. The stage of choked disk must naturally be preceded by the earlier and milder stages of disk blurring and papillœdema

and should therefore be recognized as being more significant than being within physiological limits.[1] An interesting syndrome ophthalmoscopically is that of certain frontal tumors which may in their growth produce by direct pressure down upon the ipsolateral optic nerve a primary optic atrophy, and as the result of the increased intracranial pressure there is observed in the opposite fundus a choked disk—the forerunner of a secondary optic atrophy.

II. In brain abscess, there is a replacement and substitution of brain tissue by the purulent detritus, and thus, as in gliomatous tumors which infiltrate and replace brain tissue rather than push it aside, it is rare for definite signs of intracranial pressure to be observed in these cases, unless, as has been stated before, the ventricles are blocked, or a toxic and infective meningitis occurs from the presence of the abscess; this latter condition is of frequent occurrence in subtentorial and cerebellar abscess, but it is rare for temporo-sphenoidal abscess—the most common location of brain abscess following the usual cause—an otitis media—to produce fundal changes even though the abscess may reach the size of an orange and even larger.[2]

III. Intracranial hemorrhage and œdema following fractures of the skull with or without gross brain injury rarely produce a measurable papillœdema to the extent of "choked disks." The reason for this is obvious: unlike brain tumor, hydrocephalus, and the other intracranial conditions which enlarge slowly and thus permit the brain and particularly the medulla to adapt themselves to this increased pressure with little immediate risk, in many cases of traumatic intracranial hemorrhage and œdema in the absence of marked shock the intracranial pressure rises most rapidly, so that the compensatory mechanism of the medulla has little time to adjust its vasomotor and respiratory centers to this increased pressure, and the result in these cases is death before the development of "choked disks" is possible; if these patients could survive

[1] Only too frequently is the surgically successful removal of the brain tumor possible, and yet the patient has already been irreparably damaged by the non-recognition of its pressure signs until it is too late for a normal person to be obtained.

[2] *The Laryngoscope*, March, 1914.

this greatly-increased and rapidly-produced intracranial pressure, then a measurable papillœdema and "choked disks" would occur. In brain injuries following fractures of the skull, "choked disks" do occur in the cases of large hemorrhage which have formed slowly, usually the extradural hemorrhage following a rupture of the middle meningeal artery. In these cases of intracranial pressure of comparatively slow production, the medulla can adapt itself to the pressure, and thus death does not occur before a measurable papillœdema and "choked disks" are possible.

Within the last month, I have operated upon such a case; the "choked disks" of four diopters developed five days after the basal fracture; at operation, both an extradural and a subdural hemorrhage of large amount were removed; the subsidence of the measurable papillœdema began immediately after the operative drainage of the intracranial hemorrhage, so that fifteen hours later there was present only an œdema and blurring of all the details of the optic disks but not a measurable swelling of the disks.

It is most rare in these cases of brain injuries with or without skull fracture for an œdema of the optic disks to appear within six hours following the trauma, and particularly is this true of those patients in the various degrees of shock; the greater the shock, the less the blood pressure and naturally even though a large intracranial sinus or vessel was torn yet it would be difficult for any extensive hemorrhage to occur; just as soon as the intracranial pressure equalled this lowered blood pressure, then no more bleeding could occur because the intracranial pressure would now be equal and even higher than the blood pressure; as the patient rallied from the condition of shock, then naturally the blood pressure would become higher and then more bleeding would occur, and thus the signs of intracranial pressure, such as an œdema of the disk outlines, would now be possible. In these cases the marked signs of shock usually last about four to six hours; and again, those patients who cannot survive the condition of shock—they die within six hours after the injury.

In these cases of fracture of the skull[1] it is not so important

[1] *Journal of the American Medical Association*, May 13, 1916. Vol. lxvi., pp. 1536–1540.

in the treatment to know the site and extent of the fracture, particularly if it is a basal fracture, as it is to ascertain the presence of an increased intracranial pressure, both by careful and repeated ophthalmoscopic examinations, and also by the measurement of the pressure of the cerebrospinal fluid at lumbar puncture by the spinal mercurial manometer. If there are definite signs of a marked increase of the intracranial pressure, such as an œdematous blurring of the optic disks and twice the normal pressure of the cerebrospinal fluid and even more, and the medullary compression sign of a lowered pulse rate, then I believe an early relief of this increased intracranial pressure by means of a simple decompression operation is advisable before the patient reaches the dangerous stage of extreme medullary compression and even œdema and thus collapse of the medulla itself. If an operation is postponed in these cases until a "choked disk" occurs, then the ideal time for operation will have been lost, and it is then very doubtful whether the patient will recover; besides should the patient, having had high intracranial pressure, recover without an operation or at best a very late operation, then the danger of post-traumatic conditions is very great indeed; these conditions, due to a prolonged increase of the intracranial pressure, are persistent headache, an emotional instability of either the excited or the depressed type, mental and physical lassitude and easily fatigued, and in rare cases even epilepsy in its various forms.

I feel therefore in patients having head injuries that it is most important to establish the presence or absence of an increased intracranial pressure, whether that pressure is due to hemorrhage or cerebral œdema, and that repeated ophthalmoscopic examinations are of the greatest aid in facilitating an accurate diagnosis and the early treatment of the condition.

IV. In selected cases of cerebral spastic paralysis in children due to a hemorrhage at birth, the ophthalmoscopic examination is of very great importance in aiding the differentiation of this type of intracranial lesion from the other causes of cerebral spastic paralysis. It is very interesting to note that Mr. W. J. Little, in his first monograph in 1843 upon spastic paralysis, entitled "Deformities of the Human Frame,"[1]

[1] *The Lancet*, vol. i., p. 350, December 16, 1843.

or the now so-called Little's Disease, stated that the condition was due to an impairment of nerve tissues resulting from their lack of development and also to an earlier meningitis; a few cases, however, followed difficult labors, and undoubtedly these were in his opinion due to an intracranial hemorrhage at birth. In 1862 (nineteen years later), in his second monograph upon spastic paralysis, entitled "On the Influence of Abnormal Parturition, Difficult Labors, etc., upon the Mental and Physical Condition of the Child,"[1] he says that in his opinion almost 75% of these cases are due to intracranial hemorrhage. Recent investigation of this condition also confirms this belief that about three-fourths of these cases are due to intracranial hemorrhage at the time of birth.

In order to differentiate the three chief causes of cerebral spastic paralysis in children,—that is, first, a lack of development of the cerebral cortex or its pyramidal tracts; secondly, a meningitis and meningo-encephalitis following infectious diseases such as cerebrospinal meningitis, measles, scarlet fever, and whooping-cough; and lastly an intracranial hemorrhage, it is very important that a careful ophthalmoscopic examination should be made as early as possible; the measurement of the pressure of the cerebrospinal fluid at lumbar puncture with a spinal mercurial manometer should also be used to confirm the fundal findings of increased intracranial pressure or not. Naturally in cases of lack of development of cerebral tissues, there can be no increase of the intracranial pressure, and this is also true of those cases which have survived a meningo-encephalitis—a destruction and atrophy of cortical nerve tissue; on the contrary, if an intracranial hemorrhage has occurred, then there should be signs of an increased intracranial pressure as a result of the hemorrhage as shown by a dilatation of the retinal veins and an œdematous blurring of varying degree of the optic disks; naturally, the earlier this examination is made, the more definite are the fundal signs of intracranial pressure, whereas in the older children the ophthalmoscopic examination may reveal only a dilatation of the retinal veins with thickened walls from new tissue formation and a shallow disk cup; while the disk itself

[1] *Obstetrical Transactions*, vol. iii., p. 293, 1862.

is blurred in its details, particularly along the nasal margin, the temporal margin and even the nasal half of the disk may be obscured; in no case over one year of age have I found a measurable swelling of the disk to the degree of choked disk. If upon examining a patient having cerebral spastic paralysis the above fundal changes are noted, then the pressure of the cerebrospinal fluid should be measured at lumbar puncture in order to ascertain whether the fundal changes are due to a local condition within the orbit or are possibly within physiological limits; in the children within one week after birth there is usually blood in the cerebrospinal fluid at lumbar puncture, as was demonstrated in seventeen children upon whom I operated within three days after birth.

In my last report[1] of 954 cases of cerebral spastic paralysis up to April 1, 1916, only 26% of them—that is, only one out of every four patients examined—showed these definite signs of an increased intracranial pressure, and the spastic condition was therefore due to a hemorrhage, and in these selected cases, by a cranial operation to relieve this increased intracranial pressure upon the brain, an improvement was to be obtained; at that time I had operated upon 219 children with a mortality of 16—that is, 7%. The history of these children is very suggestive: of the 219 operated patients, only 26 were not first children; only 8 were not full-term babies; only 21 were not born after a difficult labor, with or without instruments; only 49 did not have convulsive twitchings immediately after birth; and in only 21 children was the spasticity noticed before the eighth month after birth. A permission for autopsy is obtained before operation in every patient, both private and ward, and it is by this valuable means as well as by the operative findings, that the diagnosis is verified and other data ascertained.

V. The ophthalmoscopic findings in cases of hydrocephalus depend largely upon the type of hydrocephalus—whether it is of the internal type due to a blockage of the cerebrospinal fluid in the ventricles and thus producing ventricular dilatation and its resulting high intracranial pressure, or of the external type of hydrocephalus which is due to a blockage of

[1] *New York State Journal of Medicine*, October, 1916.

the escape of cerebrospinal fluid from the general cerebrospinal canal; this latter condition rarely causes a measurable papilloedema, whereas the internal type of hydrocephalus can produce choked disks of extreme degree and its subsequent secondary optic atrophy. Fortunately, in little babies before the sutures have firmly united, the skull itself can enlarge and thus a natural compensatory "decompression" takes place so that the peripheral vision may not be impaired; a drainage operation, however, offers these children the best chance of approximating normality.[1]

Besides the conditions already mentioned there are still other intracranial lesions in which an ophthalmoscopic examination is of the greatest importance; in cases of the various forms of meningitis, as an aid in differentiating the types of apoplexy, and a most important function in so many conditions—the presence of a negative fundus.

In conclusion, I may say that careful ophthalmoscopic examinations of the fundus are of the greatest importance in the differentiation of many intracranial lesions; that the signs of moderate intracranial pressure should be recognized, and that it should be realized that choked disks occur only as the result of high intracranial pressure; that the measurement of the pressure of the cerebrospinal fluid at lumbar puncture by means of the spinal mercurial manometer is the most accurate means of determining the intradural pressure; and lastly the intelligent use of the ophthalmoscope, especially the direct method, should be much more intensively studied in the medical schools and in the hospitals than it is at present.

[1] *American Journal of Medical Sciences*, April, 1917.

REPORT OF ONE HUNDRED AND THREE SUCCESSIVE CATARACT OPERATIONS; EIGHTY-THREE IN THE CAPSULE, TWENTY WITH CAPSULOTOMY.

By Dr. CHARLES B. MEDING, New York.

IN presenting this report of one hundred and three successive cases of cataract, performed at the Harlem Eye and Ear Hospital, eighty operated by the intracapsular method as performed by Colonel Smith and sometimes known as the Indian method, and twenty by the capsular method, all with iridectomy, I cannot better express my estimate of its teaching value than by quoting my honored teacher Herman Knapp:

"In cataract statistics the greatest element of fallacy lies in the judgment as to what complication, if any, should be excluded from the count. The most reliable statistics are those in which every case is counted, and even these have their fallacies. This does not mean that I underestimate the value of statistics. All statistics are good if they are honest, but they signify no more than what the reader learns by them if they furnish him the data to judge each case for himself and to estimate its relation to the sum total."[1]

The men who have had the benefit of tuition and experience under Lt.-Colonel Henry Smith, I.M.S., at his Indian hospitals have each presented reports of their work. They have returned enthusiastic and convinced. The inspiration of the master, the multitude of cases, the unprecedented practice in a condition which, save but to few, is very limited; all these have made them certain of their position and ambitious to persuade others to adopt it. Although these reports have been received with interest and courtesy, their Indian records were insuf-

[1] *Norris and Oliver System*, vol. iii., Operations. Herman Knapp.

ficient to overcome the doubts honest men expressed, and no opportunity could quickly present by and through which the matter might be further pressed. Frequently we have been informed of our silence. Several erstwhile converts have either changed their views or so modified their practice and instruments as to seem rather to invent a new operation than to be exponents of the Indian method. Reasons for this are, no doubt, absence of the master and lack of material—reasons not cheerfully acknowledged.

So matters remained up to the publication of a résumé of the subject by Arnold Knapp,[1] he in common with others having previously described a method for rendering safe an intracapsular operation. These methods aim at the dislocation of the lens without pressure on the globe, and offer various forceps for grasping the lens by its capsule and breaking its attachments by force exerted on the lens. No one can question the ease, beauty, and sufficiency of these operations, as of all others, when the conditions suit. No one of experience should belittle their difficulties under adverse circumstances.

In the résumé, there is, it seems to me, an implied condemnation of the Smith operation, expressed in selection of argument and arrangement of conclusion rather than in words, and since the title "Present Status" might seem conclusive up to date, I feel that it is fair for me to seek an unprejudiced mind on the part of my reader by presenting the various statements minus such premise. First, in reporting opinions of nine ophthalmologists who have studied with Smith, something is made of the statement that two have abandoned the operation. Now it appears that Dr. Knapp and Dr. Clark, as the two, never reported any Smith Indian results. Secondly, Dr. Millette has not studied with Smith. The résumé then might read, "Of eight who have studied under Smith, six are still enthusiastic, use and advise the operation, and one who has not been to India, uses it in fifty per cent. of his cases."

Again the statement summing up the usage of Indian students as "fifty per cent." and "certain provisions" urges me to ask if the advocate of any method impugns his advo-

[1] A. of O., xlvi. "Present Status of the Operation for Extracting," etc. A. Knapp.

cacy by less than universal use thereof. Lastly, are there any facts or figures showing that the Smith method increases the number of poor results and failures for the purpose of obtaining better vision in some additional cases?

Experience forces me to repeat that for absence of irritation or inflammation, uninterrupted recovery, and resulting eye health the intracapsular operation is phenomenal. For me vision is better, not alone in individual or particular instances, but all the time. I do not forget the pinhole and stenopaic vision often obtained by discission. I dare to think $\frac{20}{50}$ through a good-sized pupil more advantageous than $\frac{20}{30}$ through a pinhole for average patients.

As to the method by which lenses are removed in capsule there must ever be difference of choice. The advocates of the Smith method, and the great man himself, are grossly misunderstood if any gather from their labors intolerance of criticism or suggestion. What we do feel and preach and practice is an intracapsular operation in place of the capsular as the usual, accepted, classical operation for cataract, and we believe that only continued struggle with the presenting difficulties will finally achieve the finished method. Of this movement Smith is the intensive pioneer; we follow gladly. We are therefore impatient of the criticism which is not primarily constructive. It would be enough for this day if we were able to offer no more than equality of results, so great are the accompanying values of smooth, non-inflammatory, drugless convalescence. We believe we do move.

When in 1911 Kalt gave me the first pair of what he called his correct capsule forceps I saw him operate more than a dozen cases. He used the instrument to remove the whole anterior of capsule. In my presence he twice removed the lens entire. To my questioning he admitted this result in about 10% of cases, but considered the application with such intent provocative of vitreous prolapse. In two of the cases he had vitreous prolapse, both in bad patients. I had the happiness of showing the instrument to Dr. Knapp, and wish to emphasize the fact that his use of the forceps for subluxating the lens is original with him. Following his always helpful teaching and technic I again used the forceps and regularly have them at hand. I believe his method a great contribution;

since however it is presented as safer, saner, and less difficult than the Smith, since each step of the Smith operation meets critical comparison, and since most readers have used neither, let us see how largely point of view influences the preference.

On Smith's operation we are warned against its section as large, yet for the substitute we are bidden make a section just short of half.[1] Smith advocates $\frac{2}{3}$ of circumference until operator learns to measure needs of individual lens. It is said Smith objects to conjunctival flap, because of hemorrhage obscuration. Do we not need as clear a field for application of forceps within as for use of hook without? Mention is made of difficulty or failure of blood absorption in anterior chamber in intracapsular cases, yet nowhere in one hundred operations is such condition noted. Per contra, Smith prefers the McKechnie section on purely mathematical grounds. He condemns but one section, "the too small one." A conjunctival flap turned back no more affects Smith's dislocation and expression than it does iridectomy, and cut iris often bleeds more stubbornly than cut conjunctiva and its bleeding is more bothersome.

Experience forbids me crediting any particular operation with ocular hemorrhage or iris prolapse. I am convinced that the first is due to abnormal eye conditions. As a matter of fact none of the hemorrhages in this series of cases could by any chance be connected with the operative method more than to say that had no cut been made there had been no hemorrhage. As causes of iris prolapse I believe the two most prolific are inaccuracy of section and the behavior of circulatory and metabolic functions during convalescence. The unruly patient is a third and rather important cause. Fortunately such patients are the exception.

Vitreous prolapse in Smith's operation has been feared, expected, condemned, and written over the operation like Dante's sign over hell, but in the reported cases of the substitute method there were sixteen such prolapses, at least three preceding delivery (it does not say whether on section or dislocation), two loop and four spoon deliveries, yet vision seems not to have suffered and no mention is made of retarded

[1] A. of O., "One Hundred Cataract Extractions in Capsule," vol. xliv. A. Knapp.

healing in these cases. We have been told that in the capsular method vitreous prolapse is practically unnecessary. Does experience prove it? If the slightest expression of cortical matter were regularly attempted, I believe very many prolapses would occur, possibly do, and the drop or two of vitreous is hidden by lens matter. If I am answered that remaining cortical matter is negligible, I point to the indefatigable efforts at irrigation and Herman Knapp's careful instructions as to "toilette of the eye." I ask if such half operated cases know no iritis, cyclitis, glaucoma, and tough, thick, and adherent capsules. Again, if a vitreous bead prevents smooth or rapid healing, does exuding lens matter encourage it? The truth is that at times vitreous prolapse is inseparable from the cataract operation. Either on section, capsulotomy, expression, or by uncontrollable act of patient, the man who operates will meet and must care for the unwelcome accident. An infinity of words can say neither more nor less than unbiased consideration of actual results.

As to the assistant, Colonel Smith teaches the necessity of removing and preventing all pressure on the globe. It is very necessary after the delivery of a lens in its capsule to be exceedingly careful. There can be no doubt that at such time squeezing, sneezing, and instrumental accident are prolific of disaster. To prevent this Smith trained the assistant. I cannot but feel that his good fortune in securing one or two very adaptable men made him desist from further mechanical effort. In any case, knowing the requirements we are not debarred supplying them in our own way. The trained assistant is one, Green's speculum is another, Fisher's lid hook a third, and in many cases the responsive patient is best of all. Personally I like an assistant in any case, and prefer the one I am used to.

The modification of instruments is natural and largely personal. We must seek facility, dexterity, and manual comfort. We find it in our various ways. Having been twice to the Colonel in India I am more converted to his way. Later experience often clinches those teachings of our masters of which as students we are very impatient.

Next let us consider the force. It does not take force to dislocate and express the lens in capsule, a force varying

according to need. But this force is to be directed. Proper expression of a lens rarely deeply moves the vitreous. Mere pushing here and there until something happens is not the Smith operation. How many times in our studies we have yielded the hook to the master and found that our strenuous but unsuccessful effort was because of misdirected energy and non-dislocation. Appreciation of the resistance of orbital tissues, movement of vitreous, tenacity of lens attachment, together with knowledge of the mechanics of the delivery, come slowly. We should know our limitations. To persist in a determination to complete a certain maneuver rather than to remove safely and expertly a cataractous lens is not surgery. Whether Henry Smith could succeed in the particular case, whether observers will consider the change of method a defeat for either operator or operation, matters not.

One thing is evident. The choice of operation will never be other than purely theoretical until various methods are tried. No man is justified in opinion based solely on another's work or words. His estimate is practically only equal to his ability. It is ever wisest to operate as one best can, but we should all be too modest as well as too sincere to condemn anything because of our unwillingness to master it.

Lastly as to the difficulty of Smith's method, I can only say that the application of forceps to the lens with intent to dislocate is in difficulty on a par with Smith's method, case for case. The two maneuvers demand exactly the same qualifications, a steady hand, a quick eye, and a nice appreciation of force and resistance. Considering the introduction of the rather curiously bent, sharp-pointed forceps, added possibilities of wounding iris and of infection, I cannot feel that the man who masters its use could fail of the other.

In view of much that has been written as to the simplification of the intracapsular operation, presenting suggestions and instruments with which to abolish possible difficulties, I cannot too strongly remind ophthalmologists that to perform extraction by any method, the intracapsular in particular, requires fitness of touch and a degree of experience not generally necessary in other surgery. Henry Smith has always claimed that the cataract operator should specialize thereon. An annual supply of from one to six cases cannot suffice to

instruct and practice an operator. In all humility we should remember lest the facility of the few mislead the many. To me it is inexplicable that men knowing the long and self-sacrificing toil of those who excel, should think to achieve their perfection and escape their labor. Operations upon the globe are unique in that their mistakes and accidents can neither be prevented nor corrected. The master standing near is helpless. Infinite pains, utmost patience, and constant practice in section making, iridectomy, dislocation, and extraction are not only necessary but should be volitionally compulsory. Ophthalmologists should know how to approach, expose, palpate, and handle an eye; should be familiar with instruments; should be quick to see untoward effects or uncomfortable position of speculum or grasp of forceps. Suddenness, heaviness, roughness, unsteadiness are absolute bars to the Smith operation whether arising from nervousness or habit.

To minimize the difficulties of the work or the requirements of the surgeon is a greater crime than to exaggerate them. To label each step of the intracapsular operation with what masquerade as golden rules of safety, is but to trap the unwary, sacrifice eyes, and damn the method. The golden rule has already been written. It is training and experience, and there is neither short cut nor substitute. It may seem unnecessary to preface the operation with a prayer, but it is truly dangerous to approach it as one would the plucking of an eyelash.

As to my report, the tables tell the important details. All cases were operated in the Harlem Eye and Ear Hospital, under practically the same conditions, Dr. J. J. Decker assisting. Patients regularly receive sixty to ninety grains of potassium bromide three hours before operation. Alcoholics are given their daily dram. Eliminations can be made to suit the reader. Note that in the intracapsular cases medicine was used but in twelve. Note that ocular hemorrhages were all in women. The writer has had five other hemorrhages since 1897—two in India, one in Italy, and two in the Harlem Eye and Ear Hospital. Four of these were in women and three in capsular operations.

In closing I wish as always to add my tribute of honor and affection to my teachers. To Herman Knapp, of splendid memory; to Henry Smith, yet in excelling service.

No.	Name	Age	Sex	Lens	Previous Condition	Note on Operation + = Vitreous Prolapse	Healing	Vision and Remarks
1	D. A....	52	M.	Im. r.	20/200	..	..	10/10
2	L. A.....	67	M.	Im. r.	..	+	..	10/70
4	C. B.....	75	F.	Im. l.	Glaucoma.	..	Hemorrhage 3 hours. E	Enucleation.
8	W. G. B.	53	M.	Im. r.	20/200	..	..	10/10
9	W. G. B.	53	M.	Im. l.	..	..	..	10/10
10	M. B....	68	F.	Im. l.	..	+ Small section.	..	10/10
11	B.....	57	F.	Mat. r.	..	..	Prolapse.	L. Injury 4 weeks later: hemorrhage.
12	B.....	57	F.	Im. l.	..	..	..	10/70 Very unruly patient.
13	J. B.....	55	F.	Im. l.	6/20	Capsule burst, left.	..	10/20
14	R. C.....	50	M.	Mat. r.	..	..	..	10/20
15	R. C.....	55	M.	Mat. r.	..	..	..	10/10
16	C. C.....	85	M.	Hyp. l.	..	..	..	10/10
20	J. C.....	63	M.	Mat. r.	..	..	..	10/20
21	J. C.....	63	M.	Im. l.	20/200	..	..	10/10 Returned in 4 weeks with prolapse L.
22	A. C.....	72	M.	Mat. r.	..	+	Incarceration.	10/10
23	F. de B..	60	M.	Mat. r.	..	..	..	10/10
24	M. D....	64	M.	Im. l.	6/100	+ Small section.	Prolapse.	10/20
25	M. D....	64	M.	Mat. r.	..	..	..	10/20
26	M. D....	46	F.	Mat. l.	Glaucoma.	Hemorrhage in 3 min.	..	Enucleation.
28	E. D.....	70	F.	Mat. l.	..	..	..	10/10
29	A. F.....	54	F.	Im. r.	20/200	..	..	10/10
30	L. F.....	80	M.	Mat. l.	..	..	A	10/70 Hemorrhage in ant. chamber, 8th day organized. L. P.
31	A. G.....	79	M.	Mat. r.		..	Keratitis. A	10/10
32	J. G.....	73	M.	Im. r.	6/200	+	Prolapse.	10/100

33	M. H....	63	F.	Im. l.	..	..	..		
34	R. H....	51	M.	Soft. r.	20/100	..	..		
35	H. H....	63	M.	Mat. l.	..	..	..		
36	H. H....	62	M.	Im. r.	20/200	+	..		
37	R. H....	64	M.	Mat. l.	..	..	..		
38	J. H....	78	F.	Im. l.	20/200	..	..		
39	J. H. col.	75	F.	Mat. r.	..	+ Accident removing speculum.		A	D. Patient died apoplexy, 9th day. Told time with + 16.
40	S. H....	77	F.	Im. l.	..	Capsule burst—left.		A	Discission.
41	J. K....	46	F.	Im. l.	..	..		A	
43	J. R. K..	80	M.	Mat. l.	..	..	..		
44	T. K....	70	M.	Im. r.	5/200	+	..		Left hospital 10th day. Disappeared.
45	R. K....	58	F.	Mat. r.	..	Iris cut on section.	..		
46	H. K....	80	M.	Mat. r.	..	..	..		
48	M. K....	64	F.	Im. r.	..	Small section.	Prolapse excised.		Specific optic neuritis.
49	P. K....	79	F.	Mat. r.	Myopia.	..	..		
52	H. L....	80	F.	Im. r.	..	..	..		
53	M. L....	53	F.	Mat. l.	Glaucoma.	Lens with spoon.	..		Previous iridectomy.
54	M. L....	54	F.	Im. r.	Iris Adhes.	+ Lens on needle.		E	
55	T. C. L..	67	F.	Im. r.	..	Capsule burst, removed.	..		
56	K. L....	74	F.	Hyp. l.	..	Lens and vit. on section.	Hemorrhage 2 hours.		Enucleation.
57	J. L....	50	F.	Mat. l.	..	..	..		
58	J. L....	64	M.	Mat. l.	..	Lens with spoon.	..		
59	M. McC.	71	F.	Hyp. l.	Discoid lens.	..	Bad prolapse.	F	10/100 Bad patient. Resistant throughout.
60	M. McC.	56	F.	Mat. l.	..	Capsule burst, removed.	..		
61	E. McG..	75	F.	Im. r.	..	..	..		
62	A. McG..	48	F.	Im. r.	..	..	..		Best previous vision 20/40
64	J. McN..	62	F.	Mat. r.	..	+	..		
66	R. McG.	72	M.	Im. r.	5/200	..	..		

No.	Name	Age	Sex	Lens	Previous Condition	Note on Operation + = Vitreous Prolapse	Healing	Vision and Remarks
67	R. McG.	72	M.	Im. l.	..	..	Prolapse 7th day.	$\frac{20}{70}$
68	J. M....	82	M.	Im. l.	$\frac{20}{200}$	..	..	$\frac{20}{70}$
69	M. M...	71	F.	Im. l.	..	+ Capsule b. removed.	Incarceration. A	$\frac{20}{70}$
71	M. M...	53	F.	Mat. l.	..	+	Escaped from hospital.	L. P. Alcoholic mania—transferred.
72	P. M.....	70	F.	Mat. l.	Myopia.	..	..	$\frac{20}{70}$
73	J. M.....	61	M.	Hyp. r.	..	Capsule burst, left.	Incarceration.	$\frac{20}{70}$ Refused dicission.
74	A. M....	64	M.	Mat. r.	..	..	..	$\frac{20}{70}$
75	J. M....	56	M.	Im. l.	Discoid lens.	+		$\frac{20}{70}$
76	M. M...	60	F.	Mat. r.	..	..	F	$\frac{20}{400}$
77	E. J. O'S.	52	M.	Im. r.	..	+ Capsule burst, left.	..	$\frac{20}{100}$ Discission.
82	A. R....	49	M.	Im. l.	Iris adhes.	+	..	$\frac{20}{70}$
83	W. R....	64	M.	Im. r.	..	..	..	$\frac{20}{70}$
84	R. S. ...	60	F.	Im. l.	$\frac{6}{200}$	..	..	$\frac{20}{70}$
85	H. S....	72	M.	Hyp. r.	..	..	..	$\frac{20}{70}$
86	T. W. S	70	M.	Im. r.	..	..	Hemorrhage V. 8th day	L. P.
87	T. W. S..	71	M.	Im. l.	$\frac{20}{400}$	..	..	$\frac{20}{70}$
88	F. S.....	63	M.	Hyp. r.	Glaucoma.	Post. sclerotomy.	..	$\frac{20}{100}$ Previous iridectomy.
89	S. S.....	79	M.	Mat. r.	..	..	..	$\frac{20}{70}$
90	E. S.....	66	F.	Im. l.	Glaucoma.	+	A	$\frac{20}{100}$ Myopia.
91	J. S.....	69	M.	Mat. r.	..	..	..	$\frac{20}{70}$
92	H. S.....	45	F.	Mat. r.	..	Capsule burst, removed.	..	$\frac{20}{70}$
93	J. T.....	26	M.	Soft. r.	..	..	..	$\frac{20}{70}$
94	M. T....	70	F.	Im. r.	$\frac{20}{100}$	..	..	$\frac{20}{70}$
95	E. W....	70	F.	Mat. l.	Nephritis.	..	Hemorrhage 2 hours.	Enucleation.
96	K. W....	59	F.	Im. r.	..	+	..	$\frac{20}{70}$
97	S. W....	60	M.	Mat. r.	..	..	..	$\frac{20}{70}$
98	J. S.....	58	M.	Mat. r.	..	..	..	$\frac{20}{70}$
99	J. S.....	61	M.	Mat. l.	$\frac{6}{200}$	..	..	$\frac{20}{70}$
100	W. C. R.	56	F.	Im. r.	..	..	..	$\frac{20}{70}$
101	Z. B. V..	50	F.	Im. r.	Myopia.	+	..	$\frac{20}{40}$ Had worn—15.
102	N. Y....	60	M.	Im. l.	$\frac{20}{100}$	..	..	$\frac{20}{70}$

SUMMARY OF 83 INTRACAPSULAR OPERATIONS.

Vision.

3 $\frac{20}{15}$ + 14 $\frac{20}{20}$ + 24 $\frac{20}{30}$ + 12 $\frac{20}{40}$ = 53
1 $\frac{20}{60}$ + 6 $\frac{20}{50}$ + 8 $\frac{20}{70}$ = 15
1 $\frac{20}{200}$ + 5 $\frac{20}{100}$ = 6
4 Hemorrhages
1 Death
1 Left hospital
3 Light = 9

83

14 Vitreous Prolapses
9 Iris Prolapses

Without elimination 63% + had $\frac{20}{15}$ and better.
18% + had $\frac{20}{70}$ and better.

41 Immature
34 Mature
6 Hypermature
2 Soft

39 Female
44 Male

Oldest 85 Youngest 26 Average 63

No.	Name	Age	Sex	Lens	Previous Condition	Note on Operation	Healing	Final Vision and Remarks
3	L. A.....	67	M.	Hyp. l.	Myopia.	..	A	$\frac{20}{70}$ Dicission.
5	J. B.....	61	M.	Mat. r.	..	K. Forceps.	A	$\frac{20}{30}$ Dicission.
6	F. B.....	65	M.	Mat. l.	..	.. Prolapse. Transferred to Bellevue.	A	Alcoholic.
7	E. B.....	50	M.	Hyp. r.	..	..	A	$\frac{20}{30}$
17	W. C.....	65	F.	Mat. r.	..	.. Prolapse vomit.	E	$\frac{20}{30}$ Dicission.
18	M. C.....	50	F.	Hyp. r.	Glaucoma.	+ K. Forceps.	A	$\frac{20}{40}$
19	L. C.....	57	M.	Mat. i.	..	..	A	$\frac{20}{30}$
27	D. D.....	49	M.	Mat. l.	..	K. Forceps. Prolapse.	A	$\frac{20}{70}$ Refused dicission.
37	R. H.....	64	M.	Mat. l.	..	..	A	$\frac{20}{30}$
42	J. K.....	42	F.	Mat. l.	..	.. Prolapse cut.	A	$\frac{20}{30}$
47	H. K.....	80	M.	Hyp. l.	..	K. Forceps.	A	$\frac{20}{100}$ Delayed healing 3 weeks.
50	J. K.....	60	F.	Mat. l.	..	+ K. Forceps.	A	$\frac{20}{30}$
51	J. K.....	61	F.	Mat. l.	..	+	A	$\frac{20}{70}$ Dicission.
63	J. M.....	53	M.	Hyp. r.	Ant. Leucoma	..	A	$\frac{20}{70}$
70	J. M.....	56	F.	Hyp. l.	..	..	A	$\frac{20}{30}$ Dicission.
78	E. O.....	48	M.	Im. r.	$\frac{20}{100}$	K. Forceps.	A	$\frac{20}{30}$ +
79	J. P.....	67	M.	Mat. l.	..	..	A	$\frac{20}{70}$ Old Iridocyclitis.
80	C. P.....	59	M.	Im. r.	Iritic Adhes.	.. Incarceration.	A	$\frac{1}{200}$ At. Choroiditis.
81	E. R.....	47	M.	Im. l.	Prominent eye	+ K. Forceps.	A	$\frac{20}{40}$
85	H. S.....	72	M.	Hyp. r.	..	K. Forceps.	A	$\frac{20}{30}$ Dicission.

SUMMARY OF 20 CAPSULAR OPERATIONS.

Vision.

6 $\frac{20}{10} + 3\ \frac{20}{10} = 9$
5 $\frac{20}{70} + 3\ \frac{20}{50} = 8$
1 $\frac{20}{100}$
1 $\frac{6}{200}$
1 Transferred = 3

20

4 Vitreous Prolapses
5 Iris Prolapses

Without elimination 45% $\frac{20}{15}$ and better.
40% $\frac{20}{70}$ and better.

7 Hypermature
10 Mature
3 Immature

6 Female.
14 Male.

Oldest 80 Youngest 42 Average 58 +

AN IDEAL INTRACAPSULAR CATARACT EXTRACTION.[1]

By Dr. LLOYD MILLS, Los Angeles.

A REVIEW of the more recent literature makes it evident that types of intracapsular extraction of cataracts, particularly of the senile forms, are gaining in favor among American ophthalmologists.

This greater interest undoubtedly has been stimulated by the enthusiastic and persistent claims of some of the American followers of Colonel Smith of India. The more conservative of this small group considered the Smith method too highly specialized and exacting to permit of its general adoption, while the minority, exploiting, for the most part, small modifications of the original method, broadcasted the impression that the Smith method was made safe and universally adaptable by their modification. This gospel was spread among poorly or wholly untrained men, as well as among men of experience, and the natural result of such unwise teaching was to throw disrepute upon one of the most worthy cataract operations of all time. Had the trained men been as well advised of the dangers of the method as they had been of its perfections, they would have approached its performance with the necessary reverence, but to have alarming losses of vitreous, prolapse of the iris, pupillary dislocation and prolonged ciliary injection, photophobia and lacrimation accompany or follow an operation of declared simplicity proved too great a strain on the faith of the bolder operators of the country, who promptly dropped the method and who, almost

[1] Read before the Eye, Ear, Nose, and Throat Section of the California State Medical Society at Coronado, California, April 17, 1917.

to a man, oppose it. No word has come of the results of those cases operated upon by men of little or no previous operative experience, but these results may be imagined.

The perils of the Smith operation arise from the fact that in any given case the extraction may be excessively easy or excessively difficult and it requires both skill and fine operative intuition to tell which of these situations is at hand. In the former case the lens almost rolls out under gentle pressure and a continuation of this pressure or a gross touch or movement may rupture the bulging hyaloid membrane. In the latter case, owing to shallowness of the anterior chamber, to the size or hardness of the lens, to faults of the zonule, to synechiæ, to excessively low tension and vitreous degeneration from old uveal disease, and to unusual prominence of the eyeball, the dangers of this operation become so great that the wise operator changes his procedure at once to one of the extracapsular order. The attempted application of the Smith incision to eyes with very shallow anterior chambers is undoubtedly one of the most frequent causes of failure. With much care it is possible to make the counterpuncture successfully under such conditions and, by turning the blade well forward, to avoid the lens, but in many of these cases the average operator will perform an unintentional capsulotomy and an irregular iridectomy and convert the case into that least promising form of capsulotomy operation where nearly all of the capsule, an excessive amount of cortical matter, and more or less blood remain to complicate healing and visual recovery.

The iridectomy in the Smith operation gives the apparent advantage of greater room for the passage of the encapsulated lens. This larger exit is unnecessary, however, save where the lens is too firm to be molded, while the disadvantages of the iridectomy are: (1) Frequent hemorrhage from the cut vessels with obscuration of the field; (2) the resistance of the iridic ring to the passage of vitreous both while the lens is engaged in the pupil and after its delivery is lost; (3) greater extremes of dislocation of the vitreous are possible preceding, during, and after delivery and in sudden downward movement of the eye, increasing the likelihood of loss of vitreous; and (4) dazzling and color aberrancies such as erythropsia are

much increased, these last being important considerations in lands where the sunshine is sharp and glaring.

It seems probable that the Graefe extracapsular extraction will continue to be the average operation of election because of its assured safety even under most of the conditions which make any intracapsular operation yet devised unsafe or impossible. Excluding those cases in which an intracapsular extraction is manifestly or probably unwise, as in cases of high pulse pressure with a history of retinal hemorrhage, in cases where visual power remains in but one eye, subluxated lenses, and possibly high myopia, we are left to consider a large group of cataracts, mainly senile, which should be capable of intracapsular extraction. Probably the percentage of failures in any intracapsular method, as in any other major surgical measure, will depend as much upon the operator's judgment as to the method to be employed, as upon his skill, and the size of the eye, its position in the orbit, the depth of the anterior chamber, the rigidity of the pupil, and the maturity of the cataract are all factors of importance in forming such judgment. From general surgical experience, it seems doubtful if any one method of extraction will be devised equally suitable to all conditions, and my object in presenting this modification is to add to the methods of intracapsular extraction a procedure of distinct cosmetic and visual value in appropriate cases when performed by competent men. I wish it understood that this operation is by no means a simplification of the Smith method, for it requires fully as much, if not more, technical skill, but when successful the results are as near perfect, cosmetically as well as visually, as it is possible to achieve.

In brief, the method consists of a typical or enlarged Smith incision, the incision, being enlarged downward by scissors to any degree if this enlargement seem indicated at any time during the operation. The lens, in its intact capsule, is molded through the intact pupil slowly and cautiously, delivering pressure being applied through the cornea by a hook, as in the Smith method, or, in trustworthy patients, through the lower lid by finger-pressure. The latter mode of pressure, in selected cases, is most satisfactory because of the nicety ot its control. Prince's upper lid retractor and Fisher's lower lid

retractor are held by a competent assistant. As lens and vitreous cannot occupy the same place at the same time, once the lens engages the pupil and begins to mold, there is no possibility of vitreous loss until delivery of the lens is assured. After delivery of the lens, the pupil may become small and central at once, but is usually somewhat displaced upward, mainly the effect of the pyramidal dislocation of the vitreous behind the upper part of the iris. If eserine is now instilled in a 1% solution, the pupil soon becomes small and central and the immediate result so perfect that one would be tempted to end the procedure at this point. The late results of simple intracapsular extraction, however, show that more must be done for, owing mainly to the persistence of the upward and forward dislocation of the vitreous, broad retraction of the upper half of the iris and upward dislocation of the pupil are not infrequent, ciliary injection, with more or less photophobia, may last for several months, and, at times, the squeezed and distorted ciliary body shows its distress by the formation of more or less evident precipitates upon the hyaloid membrane. To prevent this, the method was adopted of instilling 1% eserine on the table for three instillations at five-minute intervals, or less, according to the fullness of its effect. The flattening of the iris, thus produced, assisted in the more extreme cases by gentle traction forward and upward upon the lower flap, aids materially in molding the vitreous back into place, and with this accomplished, a peripheral iridectomy is done above, the fineness of which is regulated by the size and prominence of the globe, the docility of the patient, and the ease with which slight subduction may cause gaping of the wound. In small and not prominent eyes only the finest possible excision is necessary to equalize tension in the chambers while in the fuller and more bulging globes a moderately broad and horizontally placed excision is necessary to prevent prolapse and pupillary distorsion.

I was led to the introduction of the method by the following considerations.

1. The ease of simple extracapsular extraction.
2. The occasional delivery of the lens in its intact capsule when attempting capsulotomy in senile cataract.
3. The ease of delivery of the lens in its capsule through

the intact pupil in experimental work on pigs' and kittens' eyes.

4. The distinct value of the fine peripheral iridectomy of Chandler—more commonly known as the Hess-Pflüger modification—in equalizing the pressure in the chambers and thereby lessening the liability to prolapse.

The beginnings of the method are shown by a sketch of the salient points of the first four consecutive and unselected cases:

The first, a man of 65, presented a mature senile cataract with a deep anterior chamber and a fairly rigid iris. After an incision of the Smith type pressure was applied to the cornea by the Smith hook to rupture the zonule and deliver the lens. Little by little this was seen to mold, to conform to the pupil, to engage, and finally to emerge to just beyond its equator. At this stage pressure was gently withdrawn and the encapsulated lens was lightly lifted out of the anterior chamber by Kalt's forceps. Not a drop of vitreous was lost. The pupil at once became small and so central that no reposition of the iris seemed necessary. 1% eserine salicylate was instilled once a day through the least possible aperture between the lids. A second patient, a woman of 89 with a hypermature cataract and a deep anterior chamber, was operated upon two days later, the encapsulated lens being easily extracted through the intact pupil and not a drop of vitreous being lost. Here also the pupil immediately became small and central and no reposition of the iris seemed indicated. At the end of the fourth day in the first case and the fifth day in the second it was noted that the pupil was becoming dislocated upward and inward and in the second case the wound bulged slightly. Eserine was increased to three times daily in the first case and a pressure bandage was applied in the second. The ectasia of the wound was easily overcome in the latter case, yet in both the pupillary displacement advanced to the final point where the middle eighth of the upper margin of the pupil was drawn just above the margin of the broad arcus senilis. Little reaction occurred in either case and none at all persisted. Vision in the first case was $\frac{6}{9}+3$ with $+9.25 = +3.50$ ax. 145 and Jaeger 1 with the reading correction. In the second case, although the media were crystal clear and there was not the least obstruction to axial vision, only $\frac{6}{20}$ and Jaeger 2 with difficulty were obtained, owing to a moderately advanced optic atrophy, not ascertainable earlier because of the maturity of the cataracts in each eye.

Having shown that simple intracapsular extraction offered no especial dangers, an attempt was made in the next case, one of traumatic cataract in a middle-aged man, to improve the cosmetic result by a more liberal use of eserine, this being instilled three times a day, with the most extreme precaution against pressure upon the globe and undue movements. While the visual result with correction was $\frac{6}{9}+$, the iris was adherent to both angles of the wound, making a horizontal ellipse of the pupil. Manfestly then eserine alone was not sufficient to prevent the gradual retraction of the iris and the application of a peripheral iridectomy to the problem was decided upon. A study of the method on the eyes of suitable animals showed that the upward and forward dislocation of the vitreous was the principal factor in producing a bulge of the iris and of the wound, and that its persistent upward lift aided materially in producing the upward retraction of the iris. The cause of this dislocation of vitreous was partly the pressure of delivery of the lens, but the greater factor and the persisting one was the new shape assumed by the globe after the lens had been delivered, the pull of the inferior rectus being practically unopposed by the superior, while the lateral pressure exerted upon the globe by the internus and externus led to a relative lateral flattening of the eyeball and a consequent realignment of its contents in a relatively sagittal plane, the bulge appearing at the point of least resistance. As the muscular factor could not be overcome save by enlarging the corneal incision downward to the point where the lateral recti became ineffectual for harm, a measure recently undertaken, the alternative measure was the reduction of pressure upon the iris by equalizing the pressure behind and in front of it. Experiment showed that the peripheral iridectomy accomplished the purpose in most cases, although where the displacement of vitreous was considerable the best result was gained by deliberately opening the hyaloid at the time of the iridectomy and permitting the escape of a few drops of vitreous. Accordingly, in the fourth patient, a woman of 68 having a mature senile cataract and a moderately deep anterior chamber, after successful delivery of the encapsulated lens through the intact pupil without rupture of the hyaloid, a fine peripheral iridectomy was placed a little to the nasal side of the vertical meridian, 1% eserine instilled at once and twice daily for two weeks. When the eye was first inspected, on the ninth day, it was difficult to tell from mere inspection which eye had been operated upon. The pupil was small, central, and mobile, the wound was flush with the cornea, the small iridectomy opening was patent, and the operation had fulfilled the highest

hopes. Final vision was full $\frac{6}{6}$ and Jaeger 1 with +10.00 =+2.50 ax. 165 and a +3.00 addition.

The method has been applied successfully to 28 cases selected from 61 operable cataracts out of a total of 89 cataracts seen during the past fourteen months and with results which justify the presentation of this paper. The exact limits or indications of the procedure are still under investigation for in over half the cases the work has been too recent to give the final visual results. Under investigation also are (1) the effects of substituting a horizontal iridotomy for the iridectomy, (2) the application of this form of intracapsular extraction with the combined peripheral iridectomy to cases where the extent of the corneal incision excludes the lateral recti from acting upon the ocular content, and (3) the use of a full peripheral corneal incision to replace the Smith incision in suitable cases.

Summary and Conclusions

1. Intracapsular extraction of cataract by any method yet devised should be performed only by men of large and constant surgical experience and possessed of keen surgical intuition.

2. It is as irrational to declare that intracapsular extraction is the only satisfactory method of removing cataracts as it is to claim that only one fixed operative procedure is applicable to the surgical cure of appendicitis or any other major surgical condition.

3. In selected cases of the Smith intracapsular extraction the final visual results are ideal. The cosmetic results are often less pleasing because of frequent pupillary dislocation. The routine application of any intracapsular method yet devised to unselected cases of cataract will bring sure disaster.

4. In selected cases extraction of the encapsulated lens through the intact pupil is a simple procedure in skilled hands. The pupil, however, is often strongly drawn upwards as a final result, although this seldom interferes with the final visual result.

5. Prolapse of the iris, bulging of the wound, pupillary dislocation, and loss of vitreous are the direct result of dislocation of the vitreous, which, in turn, is partly the result of the

pressure applied in delivering the lens and, to a far greater degree, the result of a relative lateral flattening of the globe due to a persistence of the pulls of the lateral recti, while the vertical recti are no longer in opposition owing to the (mechanically) horizontal wound in the cornea.

6. Vitreous dislocation and its results can be prevented either by so enlarging the corneal incision that the lateral recti are for the time being made as mechanically ineffective as the vertical recti or by the use of a peripheral iridectomy at the place of greatest vitreous pressure, or by both procedures combined.

7. In selected cases of cataract, ideal cosmetic as well as visual results follow the extraction of the encapsulated lens through the intact pupil when combined with a peripheral iridectomy, the extent of which depends upon the amount of vitreous displacement. Eserine salicylate in 1% solution is instilled on the table and, with appropriate care, twice daily for ten days to two weeks.

8. In the final result of this form of intracapsular extraction the pupil is small and central, the iris is mobile, the eye is protected from excessive flooding with light, and often, save on deliberate examination, the operated eye cannot be told from the unoperated one.

SOME NOTES ON BLOCKAGE OF THE TREPHINE OPENING AFTER THE ELLIOT OPERATION.

By Dr. J. W. STIRLING,

Professor of Ophthalmology, McGill University, and Oculist to the Royal Victoria Hospital.

THE blockage of the trephine opening as a sequel to the Elliot operation for glaucoma is not of common occurrence.

In forty-six operations of my own it developed in three. I have the pathological and microscopical report of only one case which came under my service during my absence from town and was operated on by one of the assistants.

Of the causes of this blocking Colonel Elliot reports dislocation of the lens, the trephine wound being made too far back in the ciliary region, this of course favoring blockage by the uvea or by the vitreous following partial rupture of suspensory ligament, and lastly hyperplasia of the subconjunctival connective tissue and iris tissue.

Of only the last two conditions can I speak from personal experience.

In one case an Elliott trephining had been done one year previously elsewhere but was followed by recurrence of increased tension. When I saw the patient the tension was 100° by Schiotz's tonometer and the opening was blocked by organizing pigmented tissue, pointing to uveal origin, at any rate in part.

A fresh trephine opening with a peripheral iridectomy was made at another point at the corneoscleral margin, after which tension fell to 30°. There was a fairly sharp reaction and persistent pericorneal injection. After a month the tension

rose, the opening being blocked by pigmented connective tissue. The glaucoma was of the congestive subacute type.

In a second case one eye was lost nine years ago from a persistent glaucoma of the subacute type, despite repeated iridectomies and a posterior sclerotomy as advised by Priestley Smith.

The second eye developed glaucoma of the subacute type with exacerbations three and a half years ago.

Trephining with peripheral iridectomy was done; in the course of a week marked iritis developed, and in four weeks the trephine opening was completely blocked and tension rose. The conjunctival flap was dissected up and found to be firmly adherent to the very edge of the trephine opening and to the adjacent sclera; a small amount of pigmented fibrous tissue was present in the opening; there had been a plastic iritis. In ten days the opening was again blocked and a second trephining was done with a peripheral iridectomy at another point in the periphery of the cornea. Rather severe reaction followed and in three days the opening was blocked. The severe pain demanded the relief of tension, although otherwise the presence of the iritis would theoretically contraindicate an operation. A sclerotomy was then done in the equatorial region followed by cauterization of the edges of the wound as advised by Kuhnt. Despite this the superjacent conjunctiva became firmly bound down and the opening blocked; the proliferation of the subconjunctival tissue together with a plastic choroiditis was evidently the main cause of this blockage. Tension rose again but was finally lowered by the excision of a large triangular piece of sclera behind the equator. This patient had marked cardio-renal trouble and some patches of choroidoretinitis could be indistinctly made out in the fundus.

The tendency to an almost malignant glaucoma and the futility of the different types of operation on the two eyes are most striking.

No doubt in the first eye the iridectomy was followed by plastic exudation which blocked the emunctories and nullified the result. The Priestley Smith sclerotomy likewise failed in effect and a second iridectomy likewise failed, pointing to a marked tendency to plastic inflammation.

In a third case the blockage and subsequent rise in tension

did not develop until nearly a year had elapsed after the Elliot operation with peripheral iridectomy had been done. The conjunctiva here was bound down and the opening filled by a plug of nonpigmented fibrous tissue, the eye was pale, and the hyperplasia of the conjunctival and episcleral tissue was apparently the main cause of the blocking, there being but slight signs of iritis.

The conjunctival stitch has always appeared to me to be a very possible path for infection—insomuch that I now omit it if at all possible.

In all these blockage cases I had used the stitch—this is a point demanding further elucidation.

Before proceeding to the discussion of the points in these cases I will give the details of the pathological and microscopical report on the case which was admitted to my ward during my absence from town; it was operated on by one of the assistants at the clinic, but the eye was lost from blockage of the wound. The eye was blind, tension almost absolute, and the Elliot operation performed as an alternative to enucleation on account of pain.

I have to thank Dr. F. T. Tooke for the report which is of much value and is as follows:

A portion of the conjunctiva bulbi which may be regarded as the site of the former bleb is intensely injected. The superficial conjunctival cells are not appreciably altered, there being no marked œdematous changes or distinct evidence of superficial necrosis. The subconjunctival tissue is so deeply injected that the finer structure of this tissue cannot be determined. The infiltration seems to consist almost entirely of small mononuclear leucocytes, and polymorphs are not present in any appreciable number. In this infiltrated tissue are two or three venous capillaries which are markedly engorged with red cells but show no evidence of migration of lymphocytes through the capillary walls in the specimens examined. This inflamed area of conjunctiva ceases abruptly at the corneal margin, the infiltration not extending in between the lamellæ of the substantia propria to any appreciable degree.

What was formerly the trephine hole is completely blocked by an infiltration of leucocytes comparable to those noted in the inflamed subconjunctival tissue as well as by inflammatory

connective-tissue cells. In this site one may distinguish a quantity of disintegrated pigment cells and one or two small capillaries which manifest the same intense engorgement as noted in the conjunctiva.

There is an intense degree of iritis, the iris being practically plastered down to the inner margin of the cornea at the filtration angle adjacent to the former trephine hole. Lymphocytes may also be seen about the ciliary processes and the ciliary body itself shows a manifest degree of inflammation.

There is, moreover, a further and considerable degree of exudate of lymphocytes thrown out about the posterior surface of the lens. There are also one or two large hemorrhages in the vitreous in the neighborhood of the posterior lens capsule, between it and the retina which is completely separated.

The lens is cataractous. From the specimens examined the process seems to have originated with a plastic iritis, or iridocyclitis, the inflammatory changes in the conjunctiva and in the corneal fistula manifesting the same histological characteristics. Although specimens were not stained for microorganisms one can hardly imagine that such agencies were directly responsible for the inflammatory changes which have occurred either in the conjunctiva or in the trephine opening.

In all of my cases the strictest antiseptic precautions were observed, cultures being taken before the operation, etc., so that an exogenous infection was not likely.

The inflammatory reaction was more of the nature of a traumatic affection associated with or favored by some dyscrasia, a so-called autotoxæmic condition, and as bearing this out two of the cases were trephined twice and each time a similar blockage occurred.

The marked plastic iritis which developed would appear to be quite distinct from the mild form of iritis observed in many cases within a day or two of the operation and which rapidly disappeared.

It would appear to be similar to the tenth day form of iritis met with after cataract extraction. It is associated with or sets up severe inflammation of the subconjunctival tissue localized to the area of the trephine opening. The deferred occurrence of this iritis would account for the lateness in the development of the blockage amounting to three or more weeks.

In Case No. 2 a development of the blockage so shortly after the second trephining would be accounted for by the iris being still in an inflamed condition, an inflammation which doubtless extended to the whole uvea as evidenced by the speedy blockage of the later trephine opening far back in the sclera, cauterization of the opening failing to prevent it.

Wounding of the lens or making the trephine opening too far back could not be advanced as a cause in any of my cases. The cause of the blocking has hence to be sought in the origin of the inflammation of the iris, associated with that of the subconjunctival tissue. Whether it be an autotoxæmic condition lighted up by the operative traumatism, or a germ infection, which latter does not appear likely, has to be further investigated although I am in favor of the former.

A CASE OF UNILATERAL VERTICAL NYSTAGMUS ACQUIRED IN ADOLESCENCE, AND CAUSED BY AN ACCIDENT.

By Dr. ANTON LUTZ, Havana, Cuba.

NYSTAGMUS has been the subject of careful research and observation in recent years. The otologists as well as the neurologists have helped the ophthalmologists in making it a more important symptom.

Pedigrees of real *hereditary nystagmus* (produced by alterations of the germinative plasma) have been rarely published, viz.: Frankenberg (Goettingen, 1909), Kibort (Zurich, 1910), Clarke and Nettleship (England, 1911), and Nodop (Leipzig, 1914). The majority of the cases of nystagmus must be considered as acquired, and these may be subdivided into those dependent upon affections of the brain, and those produced by peripheric changes of the visual apparatus. The latter are almost always acquired in the first years of life. Grimsdale tells us (*Royal Society of Medicine*, London, 1914), that every severe injury of the cornea up to the fifth year produces nystagmus. On the other side, nystagmus of peripheric origin and acquired in adolescence seems to be so rare, that Morax writes in 1905, in his "Ocular Semiology" (*Encyclopédie française d'ophtalmologie*): "On n'observe jamais chez l'adulte le nystagmus consécutive à une lésion oculaire." Wagenmann does not mention any case of nystagmus following trauma in his big monography: "Die Verletzungen des Auges mit Berueecksichtigung der Unfallversicherung" (*Graefe-Saemisch*, 1910).

In almost all these cases of nystagmus, both eyes show the same form, direction, extent, and rapidity of tremor; in only very few cases is there any difference. Holmes Spicer (*Royal Society of Medicine*, London, 1914) found in two hundred

bilateral cases the nystagmus to be in 50% horizontal, in 15% rotatory, in 12% vertical, in 4% mixed, in 2% irregular, in 2% circumductory, in 1% condivergent; the existence of the last form Ohm (1913) proved by rhythmic changes of the pupillary distance in a cinematographic film. Holmes Spicer also found that nystagmus was more influenced by darkness and by light in cases of obstruction of the media than in cases of retinal disease.

Observations of ***unilateral nystagmus*** are still rarer. P. Schmidt (Rostock, 1914) brought together all the unilateral cases published up to that date, and found only fifty-four in the whole literature, to which he added two observations of his own, from the clinic of Peters. He found that all the fifty-four unilateral nystagmus cases were not all pure cases, in so far as many showed a very slight almost imperceptible nystagmus of the second eye. Duane finds that unilateral nystagmus is mostly vertical, only seldom rotatory or horizontal. The most common form of acquired nystagmus is that of the miners and it is characterized by dancing of all objects in the field of vision, in unison with the movements of the globes. If such a case is unilateral, its form is more pendulum-like and less jerky, and it is mostly localized in a damaged eye (Coppez). This form of acquired and occupational nystagmus is now explained by the bad illumination under which coal miners have to work; the workers cannot use their cones (foveal-fixation) but only the rods so that they are in the same condition as the amblyopic. Coppez (***Société française d'ophtalmologie***, 1913) found that in cases of amblyopia the nystagmus could be: (*a*) constant; (*b*) present only when the patient tried to fix an object; (*c*) produced and checked at will. The form in most amblyopic cases was jerky (***à ressort***), sometimes irregular, seldom pendulum-like.

E. Mangold and A. Lōwenstein produced experimentally in rabbits pure unilateral nystagmus (jerk) by cutting the pes pedunculi cerebri of the contralateral side (***Klinische Monatsblätter für Augenheilkunde***, 1913).

The extraordinary rareness of unilateral nystagmus justifies the publication of the following:

C. C., 39 years old, female, white race, came to my office

for a simple conjunctivitis. Accidentally I observed her nystagmus; she told me that in the hereditary antecedents of her father as well as in those of her mother there were no known cases of nystagmus and that out of four children in two marriages, no one showed ocular tremor. Her father died very early, she does not know from what disease; her mother died of phthisis, and one son from chronic enteritis. She herself does not remember any illness, but her mother has told her that she had very severe trouble with her eyes in early childhood. When she was thirteen years old, some brick dust entered her left eye. When the bandage was removed, she was surprised to see all objects dancing before the left eye, a thing she had never seen before. Dancing of objects before the left eye has kept up except that the trouble is not so annoying as before.

Right Eye: There exists a very slight almost imperceptible nystagmus, pendulum-like, the excursion is very small, and the direction rotatory. It is not always possible to detect it, and there is no false movement before this eye when closing the other one.

Vision: $\frac{4.5}{18.0} + 1.5 = \frac{4.5}{12.0}$

Visual Field: Normal in the periphery, with small central scotoma for colors.

Pupillary Reaction: Normal to light, accommodation, convergence, and trigeminus irritation.

Media: Transparent.

Fundus: Disk, snow-white, sharply limited, vessels very narrow. There is great contrast between the aspect of the disk and the good vision.

Left Eye: Even at one meter, marked nystagmus can be seen—the form is pendulum-like, the direction vertical the extent 2 to 3*mm*, the number of oscillations two to three to the second. This eye constantly perceives false movements, which are stronger when the patient is excited. The nystagmus is best seen in the primary position, but it is also very marked in the terminal positions and does not disappear in any direction. It is constant and cannot be checked at will,— nor does it disappear during sleep and is not accompanied by movements of the head. There exists no blepharospasm, but there is strabismus, either convergens, or divergens in looking at a short distance. By stronger sensory irritation (stronger illumination) nystagmus is accelerated; on diminution of the sensory irritation by cocaine, the tremor is slower. Nystagmus does not disappear during convergence, and the patient can observe it herself in a looking-glass. She is always conscious of the false movements, and in the examination for diplopia

with a red glass, a light point appears in the form of a vertical line, never as an ellipse. Covering the right eye and forcing the left eye to work, the nystagmus does not disappear as it did in one case of Alfred Graefe; covering the left nystagmic eye, no nystagmus appears in the second one, as Neustaetter observed in his case.

Vision: < 0.10, glasses do not improve.

Visual Field: Periphery normal for moving object and colors.

Pupillary Reaction: Normal to light, convergence, accommodation, and irritation of the trigeminus.

Media: In the center of the cornea there is a superficial but faint opacity.

Fundus: Disk snow-white, sharply limited, vessels very narrow.

Motility of the Eyes: Monocular normal. Binocular defective on convergence.

This patient in her earliest life suffered from a severe inflammation of the optic nerves (of which she has no recollection) which left both optic nerves atrophic, diminished vision, and a central scotoma for colors in the right eye. This is also responsible for the slight, nearly imperceptible, nystagmus of the right eye. Sauvineau (*Encycl. franç. d'opht.*) has seen many cases of nystagmus after congenital optic atrophy. The traumatism in her twelfth year of age, producing a corneal opacity in the left eye, still further reduced the left vision. This new injury apparently provoked an unilateral nystagmus in predisposed eyes. It is very remarkable that the false movements before the eye could last all of the twenty-six years and that no divergent strabismus developed, notwithstanding the loss of vision, and that the visual impression of the left eye was not inhibited.

In all cases of nystagmus produced by corneal opacity and acquired in the first years of life the false image is soon inhibited.

A very similar case is published by Alfred Graefe (*Graefe-Saemisch*, 1898):

A man, 32 years old, anæmic and staggering in his movements, had had when 5 years old meningitis and had been treated in his college-time for hemeralopia. He shows no strabismus, but has defective vision: R. E. = 0.10; L. E. = 0.25 and marked atrophy of both disks with very narrow vessels. The right eye without any corneal lesion presented a vertical pendulum-like nystagmus, which the patient can

observe in a looking-glass. He sees no false movements. The nystagmic movements can be checked in terminal positions after he has drunk some beer.

Another similar case is recorded by Morax (Simon) (*Encyclop. franç. d'opht.*), who observed a child aged 14 months with a bilateral nystagmus after keratitis; the corneal opacity disappeared in one eye together with the nystagmus of that side, and the nystagmus disappeared finally also on the second eye together with the corneal opacity of that side. In this case the nystagmus may be regarded as a movement to enhance vision, but it is impossible to do so in my case. The nystagmus in my case must be considered an unavoidable consequence and not as an intended movement of correction.

Arlt was of the opinion, that the nystagmus would produce an enhancement of the peripheric images, which have to replace the usually distinct foveal images; this agrees with the old fact that a moving object can be seen in the periphery easier than a stationary one. In favor of this explanation of nystagmus speak also the cases of total color-blindness. There are cases with marked diminution of central vision in nicotine-alcohol amblyopia, which are never followed by nystagmus, and there is no doubt that the majority of high myopias and hypermetropias, with or without astigmatism, are not complicated with nystagmus, notwithstanding the fact that the loss of central acuity is acquired during the time of development. Such cases complicated with nystagmus are to be considered as an exception of the rule. The explanation of Arlt is even less true for the cases of unilateral nystagmus. The majority of the cases with unilateral diminution of the foveal function show no nystagmus, such as cases of macular traumatism acquired in youth, of unilateral retrobulbar neuritis, and unilateral aphakia with a healthy second eye. Krauss observed a case of nystagmus after an unilateral cataract operation, but I think such a case is an exception to the rule. It is therefore necessary to accept in all these cases complicated with nystagmus certain unknown predisposing influence as suggested by Sauvineau and Bielschowsky which make it possible for a nystagmus to develop. The optic

nerve atrophy acquired in the early life of my patient could be considered as such a predisposing influence.

Beard has taken up in his *Ophthalmic Semiology and Diagnosis* the old theory of Arlt, giving it a new aspect. He makes the comparison with a tracing paper and shows that it is easier to recognize printing through a tracing paper when it is in motion. But it is not the movement of the tracing paper which makes the object more distinct, but the unequal thinness of all these papers; one can very easily be convinced on this point by moving the paper very slowly. The comparison with the tracing paper applies especially to cases where there are darker and lighter spots in the corneal opacity. These spots are situated somewhat above and below the corneal center, which may explain the vertical direction of the nystagmus. I cannot believe that the nystagmus in my case is an intentional correctional movement, because it is not in proportion to the increase of muscular effort, and it is in disaccordance with the fact that the patient would surely feel better without the annoying false movements.

Résumé: An optic nerve atrophy acquired in earliest life has produced a diminution of vision and a central scotoma for colors. A corneal injury of the left eye in the twelfth year causes an opacity of the cornea and a consequent unilateral nystagmus; the nystagmus has persisted without any change for twenty-six years and produces annoying false images. The nystagmus is unilateral, vertical, pendulum-like, more pronounced in the primary position than in the terminal positions; the extent is 2 to 3*mm*, the number two to three per second. Squint exists at the same time, when looking at a short distance. The nystagmus cannot be considered as a factor for intentional increase of vision, but only as an unavoidable and annoying consequence of an accident.

Finally I take pleasure in thanking Professor Carlos Finlay for kindly placing his ample library at my disposal.

REPORT OF THE PROCEEDINGS OF THE SECTION ON OPHTHALMOLOGY OF THE NEW YORK ACADEMY OF MEDICINE.

By Dr. LEWIS W. CRIGLER, Secretary.

GEORGE H. BELL, M.D., CHAIRMAN. MONDAY EVENING, MARCH 19, 1917.

Dr. WILLIAM SHARPE read a paper on "The Pressure Signs of Certain Intracranial Conditions Observable in the Fundus of the Eye." This paper appears in full in this issue.

DISCUSSION: Dr. WALTER E. LAMBERT said that the importance of eye examination was recently emphasized in a case of head injury. The patient, a man fifty-five years of age, fell, striking the back of his head; he was stunned for a few moments only, and for weeks went about his business complaining, however, of severe headaches. Six weeks after the accident, the patient consulted Dr. Lambert on account of an obscuration of his vision which he had noticed for some time.

Ophthalmoscopic examination revealed a well-marked papillitis in each eye. X-ray showed a fracture through the occipital bone. Aphagia and hemiplegia of the right upper extremity developed while under observation, but cleared up within a week from time of onset. The papillitis also gradually subsided so that at the present time the fundus is nearly normal and vision is restored to $\frac{20}{20}$.

Dr. D. S. DOUGHERTY said that from the standpoint of otology, the only complications causing pressure symptoms were cerebral abscesses and, perhaps, suppurative meningitis; that while 90% of cerebral abscesses were of otitic origin, the meager statistics obtainable showed that choked disk or optic neuritis was present in but 20% of these, this being due probably, as Dr. Sharpe had said, to the fact that the pus merely took the place of the destroyed brain tissue.

He cited two cases in illustration; the first was a **post-operative mastoid** which developed obscure brain symptoms. No pressure symptoms were discoverable in the eyes; the neurologist pronounced the case one of post-operative hysteria. Post-mortem examination revealed an extensive cerebellar abscess. The second case, also a **post-operative mastoid,** left the hospital apparently in good condition with the exception of an occasional severe headache. A week or two later he was brought in in a semi-comatose condition with symptoms of meningitis, bilateral choked disks, and increase in pressure of the cerebro-spinal fluid.

Dr. MARTIN COHEN said that he had never seen a choked disk or papillœdema in the fatal or severe cases of fractured skull. He said that it is possible that death occurs too early for its development to be recognized ophthalmoscopically; and the stage of papillœdema is not reached in the cases that survive.

Dr. H. H. TYSON stated that judging from the title of Dr. Sharpe's paper he had hoped that Dr. Sharpe would give some new ocular signs whereby in certain conditions the cause of optic neuritis could be determined instead of being obliged to rely on associated signs and symptoms as at present, but that nothing new had been presented. He was of the opinion that much could be obtained by a careful examination of the retina and blood-vessels in these cases as an aid in determining the etiology.

He said that the description of the beginning optic neuritis at the nasal half of the disks and its subsequent course was the same as that given by Horsley many years ago. He disagreed with Dr. Sharpe as to all cases of papillœdema being produced only by increased intracranial pressure. He favored Gower's theory that while increased intracranial pressure alone could produce papillœdema, many cases occurred without any signs or symptoms of increased intracranial pressure, such as in anæmia, chlorosis, high hyperopia, etc.; and furthermore that in chronic hydrocephalus we rarely find optic neuritis, whereas if it was always produced by increased intracranial pressure, we would expect to find it in the majority of these cases. Dr. Tyson said that in order to produce choked disk it was necessary to have in addition to any increased intracranial pressure the

effect of the presence and pressure of inflammatory products within the papilla upon the nerve fibers and blood-vessels.

Dr. J. A. KEARNEY said that the cases of **brain tumor** seen in Dr. Sharpe's service usually have measurable papillœdema; in some instances the choked disk has resulted in secondary optic atrophy when first seen.

He said that in the selected operative cases of **cerebral spastic paralysis** due to intracranial hemorrhage at birth there was seen a measurable clear œdema more confined to the nasal margin of the disk when seen shortly after birth, but that as a rule the neurological surgeon was not called in until the œdematous swelling had begun to subside.

Dr. WILLIAM M. LESZYNSKY said that intracranial pressure is primarily due to internal hydrocephalus or cerebral œdema, or to both combined. He could not agree with the view that the pressure of the cerebrospinal fluid as measured by a mercury manometer is a reliable indication of the degree of intracranial pressure. In many instances of brain tumor with pronounced signs of intracranial pressure, such as headache, vomiting, vertigo, and choked disks, the flow of cerebrospinal fluid at lumbar puncture has been quite slow, and at times drop by drop. Furthermore, under ordinary conditions, the pressure of the fluid varies with the position of the patient and his respiratory activity. Quite commonly, when the flow is very slow it may be increased to a spurt by deep inspiration, coughing, etc.

In patients with brain injury associated with fracture of the skull, it is extremely rare to find a papillitis during the first few days. A slight degree of papillœdema, however, may develop early as a result of cerebral œdema. Later, should there be signs of intracranial pressure or secondary meningitis, a papillitis may occur. In many of these cases, great relief may often follow the withdrawal of large quantities of cerebrospinal fluid, the papillœdema disappearing as a result of this procedure.

Dr. M. J. SCHOENBERG said that examination of the spinal fluid gives valuable information as to the condition of the cerebrospinal system in cases of choked disk. He said that there was practically no danger in removing two or three *cc.* of fluid, and that the few deaths reported should not deter one

from making the examination. He does not think, however, that there is any reliable method of measuring the pressure of the cerebrospinal fluid, as there are so many conditions which alter the results, such as: blood-pressure, rate of respiration, depth of expiration, etc. He spoke of some experiments on animals by Dr. W. Parker of Detroit whereby he produced choked disk by increasing the intracranial pressure. The intraocular tension in such eyes was found to have been reduced. He thought it well for the speaker, in his abundance of material, to test out this idea.

Dr. E. F. KRUG said that Dr. Sharpe's very interesting experiments will be of real value to us if they can show us the way of early diagnosis of intracranial pressure by the kind of inflammation at the nerve head in the earliest stage of its development. It is often difficult to distinguish real inflammation of the optic nerve, and the œdema which we recognize as choked disk. Unfortunately, so many tumors of the brain develop to an inoperable stage before any eye symptoms of diagnostic value appear.

GEORGE H. BELL, M.D., CHAIRMAN. MONDAY EVENING, APRIL 16, 1917.

Dr. ALEXANDER DUANE presented for Dr. WHITE two cases: The first—a boy with a very complete **congenital paralysis of the left superior rectus,** for which Dr. White had done a tenotomy of the right inferior oblique. There were typical head tilting and a vertical deviation of 37 degrees. The result, a partial one, showed the limitations of a tenotomy done under these conditions. The deviation had been reduced by ten degrees, and the marked upshoot of the right eye had been almost abolished. As was natural with a large residual deviation there was still some head tilting. In accordance with the plan already contemplated before Dr. White operated, it was proposed to obtain further correction by advancement of the left superior rectus. The effect of tenotomy of the inferior oblique, Dr. Duane said, varied greatly in individual cases. In this case the result was insufficient, but in a similar case, with practically complete traumatic paralysis of the superior rectus, a tenotomy of the inferior oblique produced all the effect that could be desired.

The second case was that of a woman in whom a well-marked **divergent squint** with a marked upshoot of the right eye had been relieved by a tenotomy of the right inferior oblique and of both external recti. The result, both cosmetic and subjective, was remarkably good.

Dr. DUANE also showed a **plot of the field of fixation taken before and after a tenotomy of the inferior oblique in another case.** This proved that the limitation of movement produced by the operation equaled an arc of ten degrees.

As a supplement to these cases he presented a series of pictures of a patient whom he had not seen but who evidently had, as the picture showed, a paralysis of the right superior oblique with almost certainly a paralysis of the left superior rectus, associated with a marked upshoot of the right in adduction. In this case, too, tenotomy of the inferior oblique would be indicated.

Dr. WALTER B. JOHNSON presented two cases upon whom he had performed an exenteration of the orbit and covered the exposed surface with a sliding bridge flap. The first case was that of a woman, age 65, who had been suffering from a **squamous-celled sarcoma** involving both lids and ball of the right eye and extending downward on the nose. The tumor began ten years previous to operation. When seen by Dr. Johnson two years ago the involvement was so extensive and pain so severe that he decided to remove the entire affected area. This was done in July, 1915. The orbit was exenterated and the lids, together with a margin of healthy skin, removed. The skin of the right side of the forehead for a width of 2¼ inches and a depth of 5 inches was dissected free, the orbit was filled with fat from the patient's thigh, and the flap which was attached on each side was drawn over the orbital opening like a curtain. The upper margin of the flap was stitched to the periosteum of the brow and the lower margin to the skin of the cheek. The denuded area on the forehead was covered with a Wolff graft taken from the thigh. The wound was kept as clean as possible and the dressings saturated with a solution of lead and opium. There has been no return of the growth for two years.

The second case operated on by Dr. Johnson was that of a woman fifty years of age who had had an **epithelioma of the**

cheek and eyelid for four years. The operation in this case was similar to the first except that the fat and Wolff graft were taken from the abdomen. Recovery was uneventful. There has been no return for six months. Neither patient has suffered since operative recovery and their appearance is much less disfiguring than would be expected.

Dr. J. M. WHEELER presented a patient on whom he had performed Krönlein's operation for the removal of an unusual **orbital tumor.** The patient a woman, 40 years of age, was first seen in July, 1915. The beginning of her trouble dates back ten years when she first noticed a small red spot in the right eye. Later the eyelids became black and blue and from that time on the eyeball began to slowly protrude until six months previous to her first visit the exophthalmos became suddenly more marked. Examination revealed a large sub-conjunctival hemorrhage to the temporal side of the cornea. The superficial veins of the upper and lower lids were dilated. There was a decided protrusion in the region of the lacrimal gland. Movements of both eyes were normal, although there was lack of fusion. The patient's physical condition was good. Vision was not apparently affected by the growth.

On July 22, 1915, Dr. Wheeler performed the Krönlein operation. On cutting through the tarso-orbital fascia a soft tumor mass presented. This was adherent to the lacrimal gland and jelly-like in consistency. It appeared to be entirely to the temporal side of the eyeball and outside of the cone of muscles. A small area of the upper part of the temporal wall of the orbit was deficient. The tumor mass was removed as thoroughly as possible and the lacrimal gland removed. The X-ray plate shows a definite cloudiness in the right orbit. Microscopic sections of the growth were made by Dr. Geo. S. Dixon. He considered it a hemorrhagic form of degenerating connective-tissue tumor. Dr. Weeks considered it probably sarcomatous. Dr. F. H. Verhoeff took it to be an unusual form of hemangioendothelioma. Dr. F. C. Wood said that he could not diagnose the condition. All agreed that the tumor had no connection with the lacrimal gland. There is now no evidence of recurrence and the right eye is no more prominent than the left.

DISCUSSION: Dr. TYSON said that he saw this patient in

1906. He said that at that time he made a diagnosis of hemangioma with recurrent hemorrhages. He advised exploratory operation but the patient refused.

Dr. MARTIN COHEN presented a case of general muscular atrophy complicating binocular ophthalmoplegia externa with marked exophthalmos. The case was that of a Russian, male, age 46. His family history, negative; past history—excessive alcoholic until two years ago. The patient noticed five years ago that both eyes began to protrude, and he became a sufferer from severe headaches unaccompanied by vomiting. Patient has had some difficulty in swallowing, and complained of attacks of pain beneath the sternum, sometimes accompanied by vomiting which relieves him. A year ago he noticed that the muscles of his hands tired readily. Muscular weakness soon extended to his legs, and progressed to such an extent that he was incapable of remaining on his feet for any length of time.

Physical examination: The patient is poorly nourished. There is a marked muscular atrophy of the shoulder girdle, the hands, face, and lower extremities. The electrical reactions show a diminished response to stimulation in the atrophied muscles. There is no reaction of degeneration, and no evidence of cerebral lesion. Blood, urine, and Wassermann tests of blood and spinal fluid were all negative.

X-ray examination reveals an enlarged sella with erosion of posterior clinoid processes, suggesting possible pituitary disease. X-ray of the œsophagus revealed a stenosis due to the pressure of enlarged lymph nodes.

Ophthalmological Examination: Exophthalmos of right eye equals 33*mm*, left 35*mm*. Binocular ophthalmoplegia externa of nuclear origin is present. The eyes are otherwise normal. The case is of interest on account of the inability to correlate the three prominent symptoms.

Dr. T. H. CURTIN presented two cases upon whom he had operated successfully for the **relief of detachment of the retina.** The first case shown had been presented to the members of the section on two previous occasions. The purpose of again bringing the case is to show that the retina is still in place almost three years from the date of operation. The patient's vision with correction is now $\frac{20}{20}$, and his field is normal.

The second case was seen by Dr. Curtin for the first time in September, 1916. The patient a man, 62 years of age, gave a history of sudden loss of vision in the right eye in the early part of July, 1916. Previous to that he had been told by an optometrist that his vision in that eye was normal. When Dr. Curtin first saw him his vision was only fingers at two feet in the lower field. Ophthalmoscopic examination revealed a flat detachment of the entire lower half of the retina with floating opacities in the vitreous. On October 7, 1916, Dr. Curtin by means of a *2mm* trephine removed a button of sclera, aspirated 15 minims of clear yellowish fluid, and replaced the conjunctival flap. On October 8th, patient said that he could see much better. A few days later he could count fingers at eight feet. Ophthalmoscopic examination showed the retina to be in place and on October 30th he left the hospital with a corrected vision of $\frac{20}{100}+$, could read Jaeger No. 10 with +2 D. S. added to his distance correction; on November 15th his vision was $\frac{20}{200}$ with correction.

At the present time the retina is completely reattached, but there are numerous vitreous opacities which partially account for the diminution of his vision.

DISCUSSION: Dr. J. M. WHEELER asked Dr. Curtin how many cases he had thus operated and what results had been obtained.

Dr. N. L. WILSON asked if it would not do just as well to aspirate the subretinal fluid without making the trephine hole in the sclera.

Dr. CURTIN said that he and Dr. E. S. Thomson had performed the operation about sixty times with favorable results in six cases. He said that the reason for making the trephine opening in the sclera was for the purpose of permanent filtration, and besides these eyes are usually soft and the traumatism associated with aspiration might further injure the eye. He said that in the successful cases the tension returned to normal.

Dr. GEORGE W. JEAN presented a patient who has for the past ten months had a **descemetocele** which has remained flat and transparent.

Dr. JEAN presented a case of **sarcoma of the conjunctiva** which had recurred locally after removal. His reason for not resorting to a more radical procedure sooner was that the

man's other eye was practically blind. He proposes now in spite of this fact to exenterate the orbit, as the tumor shows signs of rapid growth.

DISCUSSION: Dr. H. W. WOOTTON said that Dr. Crigler, several years ago, read a paper on "Epibulbar Sarcoma," and showed by a compilation of the cases previously reported that sarcomata of the conjunctiva are just as malignant as such tumors situated on any other exposed part of the body. If such be the case it would seem wise to deal with these tumors in a radical manner in the very beginning.

Dr. L. W. CRIGLER said that he did not think it necessary to exenterate the orbit in all of these cases. He said that simple removal of the tumor alone should be confined to those cases where we are reasonably sure that there has been no extension beyond the conjunctiva; this is evident by the fact that the conjunctiva is freely movable over the site of the tumor, and a history of very slow or imperceptible growth over a period of several months. If the conjunctiva is the least bit adherent to the underlying parts, that part, whatever it is, should be removed; if it is the sclera, the eyeball, together with a wide margin of healthy conjunctiva, should be sacrificed. If the tumor is well advanced in size when first seen and there is a history of rapid growth, exenteration of the orbit should be performed at once.

Dr. ALEXANDER DUANE reported an unusual case. The patient's right eye showed degenerative changes at the macula. There was a small central scotoma. There was no evidence of general involvement of the fundus. Examination of the blood showed complement fixation for the staphylococcus albus. Transillumination over the right antrum gave a dark shadow. X-ray revealed a circumscribed mass, impervious to the rays, situated in the antrum. The patient was referred to Dr. Coakley who, on opening the antrum, found the mass to be composed of a mixture of bismuth and pus. The patient could give no clue as to its presence there. Dr. Duane thinks it was used in the treatment of a former sinusitis. He proposes to wait a reasonable length of time after the removal of the bismuth and in the event the central scotoma does not disappear, he will prepare and use a vaccine made from a culture of the staphylococcus albus.

Dr. MARTIN COHEN presented a microscopical section of an eye in a case of **spleno-myelogenous leukæmia.** A pathological report of this specimen and drawing will be presented in a future number of these ARCHIVES.

REPORT ON THE PROGRESS OF OPHTHALMOLOGY FOR THE THIRD AND FOURTH QUARTERS, 1916, AND THE FIRST QUARTER, 1917.

By H. KOELLNER, Berlin; W. KRAUSS, Marburg; R. KÜMMELL, Erlangen; W. LOEHLEIN, Greifswald; H. MEYER, Brandenburg; W. NICOLAI, Berlin; H. PAGENSTECHER, Strassburg; **K. WESSELY**, Würzburg; and M. WOLFRUM, Leipsic, with the Assistance of Drs. ALLING, New Haven; CALDERARO, Rome; CAUSÉ, Mayence; CURRAN, Kansas City; DANIS, Brussels; GILBERT, Munich; GROENHOLM, Helsingfors; v. POPPEN, Petrograd; TREUTLER, Dresden; and VISSER, Amsterdam.

Edited by MATTHIAS LANCKTON FOSTER, M.D., F.A.C.S., New Rochelle, N. Y.

I.—GENERAL OPHTHALMIC LITERATURE.

1. DALAND, J. **Eskimo snow blindness and goggles.** *Ophthalmic Record*, March, 1917.
2. JESSOP, W. H. **Ophthalmological lesions in the war.** *Archives d'ophtalmologie*, XXXV., 4.
3. MORAX, MOREAU, and GENET. **Report on the evaluation of invalidity caused by hemianopsia.** *Archives d'ophtalmologie*, XXX., 4.

DALAND (1, **Eskimo snow blindness and goggles**) states that the common ocular diseases of the Eskimo are conjunctivitis, cataract, and snow blindness. The last affection is guarded against by the use of goggles made of wood or bone with only a slit left for vision. Animals are likewise affected with snow blindness, as a bear has been observed with the trouble. One attack predisposes to another, and ultra violet rays are supposed to be the cause.

ALLING.

W. H. JESSOP (2, **Ophthalmological lesions in the war**) says that among the few ophthalmological lesions gained from the war the complete absence of sympathetic ophthalmia, the

great frequency of papillœdema in cranial injuries, and the retinal changes in trench nephritis are the most interesting. The last condition he studied, together with Foster Moore, in 15 patients and has been described in the *Lancet* December, 1915.

SCHOENBERG.

MORAX, MOREAU, and GENET (**3, Report on the evaluation of invalidity caused by hemianopsia**).

Marie and Chatelin have found that 10% of soldiers with cranial injuries have some involvement of the cortical optic centers with hemianopsia as the principal symptom. The authors attempt to establish the amount of compensation due to soldiers with such disturbances. It is usually thought that a homonymous hemianopsia (right or left), involving the halves of the visual field of each eye, is equal to the loss of one eye and requires a compensation of 30%. The authors think that this opinion should be revised. The nasal half of the field having value of 12% of the entire field of vision and the temporal half 24%, the two halves equal to 36% instead of 30%. The homonymous hemianopsia is impairing the vision much more than the loss of one eye. Regarding the estimation of compensation for quadrant hypohemianopsia, we have to figure that 6% is the value of each nasal quadrant, and 12% of each temporal quadrant.

For a bilateral hyperhemianopsia they figure, according to the values set above, 64% as the commensurate value.

Hemianopsia in an individual who has had one eye enucleated should be compensated by 36% for the hemianopsia and 30% for the loss of the eye.

For bitemporal hemianopsia they allow 64% and for binasal hemianopsia 32%.

SCHOENBERG.

II.—RELATIONS OF OPHTHALMIC TO GENERAL DISEASES AND POISONS

4. BLAKE, E. M. **Complete bilateral ophthalmoplegia with choked disks.** *Ophthalmic Record*, February, 1917.

5. BRUNER, W. E. **A case of pituitary body disease with reference to the effects of early operation.** *Trans. Amer. Ophthalmol. Soc.*, 1916.

6. CALHOUN, F. P. **Visual fields in pellagra.** *Ophthalmic Record*, February, 1917.

7. FERNANDEZ, J. S. **Ocular disturbances due to malaria and quinine.** *Ibid.*, March, 1917.

8. GREMEAUX, P. **The ocular lesions due to the action of lacrimogenic gas.** *Clinique Ophtalmologique*, vii., 10.

9. KEARNEY, J. A. **Studies of eyeground changes in cerebral spastic paralysis.** *New York Medical Journal*, February 3, 1917.

10. LANCASTER, W. B. **A case of botulism.** *Transactions of the American Ophthalmological Society*, 1916.

11. PETER, L. C. **Eye phenomena observed in three hundred and twenty cases of pulmonary tuberculosis.** *American Journal of Ophthalmology*, April.

12. POSEY, W. C. **The ocular complications of anterior poliomyelitis.** *Ophthalmic Record*, December, 1916.

13. SHARP, W. N. **A case of aniline poisoning.** *Ibid.*, November, 1916.

BLAKE (4, **Complete bilateral ophthalmoplegia with choked disks**) reports the case of a young girl who had suffered from an attack of grippe. About six weeks later she complained of severe headache and dimness of vision. There were complete blindness and total ophthalmoplegia with intense swelling of both disks. She entirely recovered after a few weeks except for pallor of the nerves. In the absence of other causes the diagnosis was made of serous meningitis due to the grippe germ.

ALLING.

BRUNER'S (5, **A case of pituitary body disease with reference to the effects of early operation**) case when first seen showed only a small scotoma for red on the temporal side of the fixation point. Later there appeared bitemporal hemianopsic scotomata for red, extending about 20° from the fixation point, and partial scotoma for white in the same areas. The nerves were slightly pale. An operation removing the floor of the sella turcica resulted in nearly complete disappearance of the defects in the fields.

ALLING.

CALHOUN (6, **Visual fields in pellagra**) has examined about forty cases of this disease and has found, especially in advanced cases, a decided pallor of the nerve. He believes also that most cases of pellagra have contraction of the field for form and colors, with frequent interlacing and misplacements of colors, especially green and red. A large proportion show red and

green scotomata. These signs are often among the first observed.

ALLING.

FERNANDEZ (7, **Ocular disturbances due to malaria and quinine**) states that malaria may produce neuralgia and hemorrhage of the retina, but that quinine, in doses over one gram, gives sudden blindness, with atrophy of the optic nerve, narrow vessels, and contracted fields. Vision may improve to some extent after the first loss. He says that degeneration attacks the cells of the retina first and later the nerve fibers and the nerve itself.

ALLING.

PETER'S (11, **Eye phenomena observed in three hundred and twenty cases of pulmonary tuberculosis**) observations were made on patients in the last stages. He noted, in practically all, an engorgement of the retinal veins due to malnutrition and cardiac weakness. He found, also, a conspicuous absence of phlyctenulosis and in no case did he find tuberculous lesions in the uveal tract. There was often a tendency toward widening of the palpebral fissure and Graefe's sign due to absorption of orbital fat and relaxation of the orbicularis.

ALLING.

KEARNEY (9, **Studies of eyeground changes in cerebral spastic paralysis**) says that the following is the usual appearance of the eyegrounds of children who exhibit spastic signs due to intracranial hemorrhage at birth. The entire fundus is slightly hazy and appears as though particles of finely ground black pepper had been shaken lightly upon it, and the color of the background is red. The temporal portion of the disk is paler than normal and its margins are plainly visible, while there is an increased redness in the nasal portion and a slight blurring of its margins, and the upper and lower margins near the vessels are blurred also. The veins are distended slightly and the light streaks of all of the vessels are either missing or barely visible. Some eyes exhibit milder signs, in others they are even more marked. A few show postneuritic signs, such as pallor of the entire disk, indistinct margins, and light streaks of exudate along the vessel walls with a leveling of the physiological cup. Some show an uneven distribution of subretinal pigment, others an entire loss of pigment. A

general œdema, or a papillœdema, may be found. Kearney urges that every child whose delivery was difficult or instrumental should have his fundus examined directly after birth, and then that if pressure signs are found, which are confirmed by the measurement of the cerebrospinal fluid on lumbar puncture, a decompression operation should be performed.

FOSTER.

P. GREMEAUX (8, **The ocular lesions due to the action of lacrimogenic gas**) found these to consist of an acute congestion of the palpebral as well as bulbar conjunctiva and a superficial desquamation of the periphery of the cornea. The condition is benign, the symptoms (lacrimation, photophobia, burning sensation, etc.) subside in a few days or weeks. The only serious complication—infiltration of the cornea—was seen in patients with previous infections of the lacrimal sac. Occlusive bandages are contraindicated, since the healing process of the cornea is very much retarded by this procedure.

SCHOENBERG.

POSEY (12, **Ocular complications of anterior poliomyelitis**) saw a case of this disease which ran a regular course with paralysis of the left arm and right leg which later disappeared. The child also complained of diplopia, and examination of the eyes showed paralysis of the left sixth nerve and optic atrophy of both eyes. The atrophy was not of the post-neuritis type and may have developed from the action of toxins. Optic atrophy is exceedingly rare in anterior poliomyelitis. Four cases were seen showing various motor paralyses. It appears likely that these paralyses are due to nuclear or intramedullary lesions rather than to meningitis.

ALLING.

LANCASTER (10, **A case of botulism**) reports the case of a woman who was suddenly taken with total ophthalmoplegia, except of the sphincter of the iris, accompanied by nausea, vomiting, and vertigo. Later there were dysphagia, disturbances of speech, and general muscular weakness. The vision was not affected. An interesting feature of the case was a changing astigmatism with constant readings of the ophthalmometer giving evidence of a lenticular astigmatism due to unequal contraction of the ciliary muscle, and this was confirmed by examination under a cycloplegic which eliminated

the error. For lack of better cause the cause was ascribed to poisoning probably by decomposed meat.

ALLING.

SHARP (13, **A case of aniline poisoning**) treated a case in which a piece of aniline dye had lodged in the eye. Small particles were imbedded in the conjunctiva and could not be thoroughly removed. There was violent reaction and areas of sloughing of the conjunctiva appeared.

Iridocyclitis ensued with hazy cornea and exudate in the anterior chamber.

At the end of six weeks the process began to subside and finally fully cleared.

ALLING.

III.—TREATMENT.

14. DARIER, A. **Treatment of intraocular hemorrhages.** *Clinique Ophtalmologique*, vii., 9.

15. FROGIER, L. **Paraspecific treatment per os for ocular infections.** *Ibid.*, vii., 6.

16. MOREAU. **Early treatment of war injuries of the eyeball and lids.** *Ibid.*, vii., 6.

17. TERRIEN, F. **The X-ray treatment for visual disturbances of hypophyseal origin.** *Archives d'ophtalmologie*, xxxv., 5.

DARIER (14, **Treatment of intraocular hemorrhages**) discusses the various methods of treatment and finds some merit in the use of radium, auto-serum therapy, subconjunctival injections, and potassic iodide. For recurrent vitreous hemorrhages in adolescents he recommends a number of well-known remedies to be used in accordance with the ætiology of the condition, such as tuberculin, thyroid extract, and antiluetic remedies. The spontaneous intraocular hemorrhages, so much feared by ophthalmologists during a cataract operation, are best treated by an aspiration of from 50 to 100*cc* of blood from a vein in the arm, and the reinjection of the fluid immediately under the skin of the abdomen.

SCHOENBERG.

FROGIER (15, **Paraspecific treatment per os for ocular infections**) renews Darier's frequent pleas for the use of antidiphtheritic serum given by mouth in ocular infections.

SCHOENBERG.

MOREAU (16, **Early treatment of war injuries of the eyeball and lids**) pleads for the utmost possible conservatism in dealing with eyeballs which have been severely injured in the war, and for the immediate care of the lacerated lids and tissues of the orbit. This point of view is responsible he says, for many an eye saved which would otherwise have been considered as beyond any hope of recovery, and also for several good cosmetic results in soldiers with extensive wounds in the face and orbit. He recommends the use of antitetanic serum in patients with even insignificant superficial abrasions of the cornea, because there are cases on record in which the cornea was the point of entry for the tetanic infection.

Regarding the treatment of severe perforating injuries of the eyeball, the author recommends irrigations of the wound twice a day with artificial serum and the application of the electromagnet. The X-ray diagnosis often is not necessary as long as the magnet is used in every case; besides, the radiography may give a negative answer when the foreign body is very small. The electromagnet applied in a routine manner is of most decided diagnostic and therapeutic value. The results in injuries of the lids depend upon how early they are taken care of, and he urges that wounds of the eyeball, lids, and face should be cared for, and attempts made to repair the tissues, at the earliest possible time.

SCHOENBERG.

The unsatisfactory results obtained by the surgical treatment of tumors of the hypophysis led Gramegna, Béclère, and Jangeas to try the use of X-rays. Terrien (**17, X-ray treatment for visual disturbances of hypophyseal origin**) reproduces in his paper the histories of ten cases (of Gramegna, Béclère, DeLapersonne, Darier, Valude, Cauvin, Calamet, Terrien) showing that in most of them headaches, diminution of acuity of vision, and the contraction of the field of vision were greatly or totally relieved by the cautious use of X-rays. The technic of X-ray application is described in Béclère's paper (*Journal de radiologie et d'électrologie*, 1914, 1°). In seven cases the headache disappeared after one single application and did not return for three years following.

The acuity of vision improved from 0 to $\frac{1}{60}$, and from $\frac{1}{10}$ to $\frac{7}{10}$, etc.

The X-ray treatment should be applied as early as possible. Cases far advanced are not a contraindication for treatment. Cases have been seen in which the treatment arrested the process or even improved fields pretty much contracted. In some patients amblyopic or even amaurotic zones of long duration have disappeared completely, this all taking place because the physiologic block has been relieved. On the other hand, in other patients parts of the field of vision have gradually disappeared during the treatment. It follows that the patients should be very closely watched.

SCHOENBERG.

IV.—REMEDIES, INSTRUMENTS, AND DIAGNOSIS.

18. AUER, E. M. **The oculocardiac reflex in syphilis of the central nervous system.** *Journal A. M. A.*, March 24, 1917.

19. CANTONNET, A. **Association of the Claude Bernard-Horner syndrome with the syndrome of Avellis.** *Archives d'ophtalmologie*, xxxv., 3.

20. CHARLES, J. W. **Dental stigmata of syphilis and transillumination in the demonstration of Hutchinson's teeth.** *American Journal of Ophthalmology*, March, 1917.

21. DARIER, A. **Arsenical preparations, their indications and method of employment; the adjuvant action of sulphur.** *Clinique Ophtalmologique* vii., 10.

22. KENNEDAY, F. **A further note on the diagnostic value of retrobulbar, neuritis in expanding lesions of the frontal lobe.** *Journal of the American, Medical Association*, November 4, 1916.

23. LANGDON, H. M. **Isolated paralysis of the cervical sympathetic accompanying goiter.** *Transactions American Ophthalmological Society*, 1916.

24. TERSON, A. **Ectogan in ocular therapeutics.** *Clinique Ophtalmologique*, vii., 7.

25. TERSON, A. **The Bergonie electro vibrator in ophthalmology.** *Archives d'ophtalmologie*, xxxv., 3.

26. WALKER, E. B. **Color interlacing and perimetry.** *Transactions American Ophthalmological Society*, 1916.

A. DARIER (21, **Arsenical preparations, their indications and method of employment; the adjuvant action of sulphur**) outlines the well-known methods, indications, and contraindications for the use of salvarsan and neosalvarsan. Optic atrophies are not a contraindication, if these drugs are used at the beginning of the process and with caution.

The greatest care and caution should be exerted with the

use of salvarsan in optic neuritis and muscular paralysis. Enesol, hectine, galyl, luargol, and various arsenical preparations are to be used with discrimination; their beneficial effect and harmfulness have not been worked out yet.

Regarding the action of sulphur, Darier is inclined to accept the views of McDonagh who is very much in favor of a preparation of sulphur called, "intramine." This drug has the property of preventing, or at least attenuating, hydrargyrism and of augmenting the antisyphilitic action of salvarsan.

SCHOENBERG.

A. TERSON (24, **Ectogan in ocular therapeutics**) has had excellent results with 1–2% of ectogan ointment in the treatment of wounds and ulcerations of the conjunctiva, cornea, and lids. Its action is as antiseptic as that of iodoform, but is devoid of its two main drawbacks: toxicity and unpleasant odor. Ectogan is peroxide of zinc.

SCHOENBERG.

A. TERSON (25. **The Bergonie electro vibrator in ophthalmology**). The electro vibrator of Bergonie is an electro magnet with an alternating current. It is of great aid for the localization of very small magnetic fragments in the eye and often supplants an X-ray. SCHOENBERG.

The phenomenon referred to by AUER (18, **The oculocardiac reflex in syphilis of the central nervous system**) is a slowing of the pulse by pressure on the eyeball. The cardiac inhibitory center is a bilateral group of cells in the medulla. The afferent impulses from the eye are carried through the fifth nerve to the Gasserian ganglion, thence through the larger root to its nucleus on a level with the middle of the superior peduncle of the cerebellum and to the inhibitory center of the vagi. From an examination of thirty-seven cases of syphilis of the central nervous system he concludes that absence of the reflex is among the earliest signs in these cases and easily demonstrated by the general practitioner. ALLING.

CHARLES (20, **Dental stigmata of syphilis and transillumination in the demonstration of Hutchinson's teeth**) uses an ordinary hand electric light to transilluminate the teeth and is able to demonstrate the presence of deficient enamel even in cases which do not show the usual notching.

ALLING.

KENNEDAY (22, **Diagnostic value of retrobulbar neuritis in expanding lesions of the frontal lobe**) describes the case of a woman whose symptoms began by dimness of vision in the right eye, enlargement of the blind spot, and central scotoma. The pupil reacted to light but immediately rebounded under constant illumination. There was later some evidence of deterioration of the motor function of the left side of the body. An X-ray showed a faint shadow in the right frontal lobe. These signs pointed to a large frontal lobe tumor pressing on the optic nerve and somewhat on the motor area. A frontal decompression was done but the patient died suddenly after a convulsion three months later. Examination of the brain revealed a ruptured aneurysm of the right internal carotid artery about the size of a walnut.

ALLING.

LANGDON (23, **Isolated paralysis of the cervical sympathetic accompanying goiter**) saw two cases, one of which had a small simple goiter. In each the instillation of cocaine produced in one eye no effect on the palpebral fissure, pupil, or protrusion of the eyeball. These signs together with the presence of hemiatrophy of the face made diagnosis of paralysis of the sympathetic probable. ALLING.

A. CANTONNET (19, **Association of the Claude Bernard-Horner syndrome with the syndrome of Avellis**). A soldier was injured by a fragment of a shell which entered the left side of the neck below the mastoid. He presented the following rare complex of syndromes.

1. The Claude Bernard-Horner syndrome (enophthalmus, myosis, ptosis, atrophy of the skin on the left side of the face, lower temperature and increased sudation on same side).

2. The Avellis syndrome (complete paralysis of the left side of the soft palate and of the left vocal cord).

3. Paralysis of the left hypoglossus and left glossopharyngeal nerves.

The author advances the idea that in paralysis of the upper portion of the cervical sympathetic we find only oculomotor disturbances (ptosis, myosis, enophthalmus), while in lesions of the lower portion of the sympathetic we find also disturbances of the sudorific glands and of circulation.

SCHOENBERG

WALKER (26, **Color interlacing and perimetry**) calls attention to the various psychological, physical, and mechanical errors which are liable to vitiate the results in field-taking especially with colors, and describes the method which he employs to eliminate such faults as much as possible. The phenomenon of color interlacing, especially in cases of intracranial pressure, was at one time thought by certain observers to be quite pathognomonic. The author, however, has come to the conclusion that the test is not reliable and that the results obtained were due to insufficient cognizance of the many subtile errors of observation. His experience has been wide, as shown by the accompanying table of cases examined.

ALLING.

V.—ANATOMY, EMBRYOLOGY, MALFORMATIONS.

27. WRIGHT, H. R. **Anophthalmos.** *Ophthalmic Record*, December, 1916.

WRIGHT (27, **Anophthalmos**) saw an infant, which otherwise appeared to be normal, with the eyes represented only by small pigmented spots, from which numerous blue-black lines radiated. No rudimentary eyes could be felt on palpation.

ALLING.

VI.—THE SENSE OF SIGHT.

28. BARGY, M. **Remarks on reversed vision.** *Clinique Ophtalmologique*, vii., 10.

29. LANDOLT, E. **The visual acuity at short distances.** *Archives d'ophtalmologie*, xxxv., 4.

30. VINSONNEAU. **The central vision in hemianopics with the macula left intact.** *Ibid.*, xxxv., 5.

BARGY (28, **Remarks on reversed vision**) has seen a patient who told him that during a severe attack of migraine words appeared reversed. The attack lasted a few seconds. He calls attention to the fact that the French writer George Sand described in her memoirs a similar attack of reversed vision.

SCHOENBERG.

LANDOLT (29, **The visual acuity at short distances**) says that the visual acuity at short distance is not in direct proportion with the one at long distance. The clear vision at

short range depends on the static and dynamic state of refraction of the dioptric system of the eyes. The size of retinal images and the acuity of vision change for the same visual angle when the eye is looking far or near. The image and the visual acuity are greater in near vision. The author has figured and tested out practically a chart of optotypes to be used for examining the acuity of vision for near and maintains that the results obtained justify in testing the visual acuity in all eyes, in a routine manner, for near with his optotype chart.

SCHOENBERG.

VINSONNEAU (30, **The central vision in hemianopics with the macula left intact**) has noticed that in a large percentage of cases of traumatic injuries of the occipital region with homonymous hemianopsia the acuity of vision in the two eyes was not equal (refraction, fundus, media being equally normal): the vision was more reduced in the right eye if there was a right-sided homonymous hemianopsia, and vice-versa. There are fourteen such cases in the French literature and the author reproduces the histories of two of his own patients illustrating this condition.

An explanation for the difference in acuity of vision in such cases may be found if we accept the hypothesis that there is a difference of size of the two macular bundles (straight and crossed) which originate in the occipital lobe and go to each eye.

SCHOENBERG.

VII.—THE MOTOR APPARATUS OF THE EYES.

31. ALGER, E. M. **Paralysis of divergence.** *Transactions American Ophthalmological Society*, 1916.

ALGER (31, **Paralysis of divergence**) has seen nine cases of this sort. The characteristic signs are full motility in lateral motion, a convergent strabismus which is present because the function of divergence is absent, and homonymous diplopia for distance which decreases as the object of fixation is brought nearer, and disappears at a certain point of equilibrium. The diplopia decreases when the patient looks to either side,

and the deviation behind a screen is equal in the two eyes, which is not the case in true motor paralysis.

ALLING.

VIII.—LIDS.

32. DORE. **Involuntary movements of the upper lid.** *Clinique Ophtalmologique*, vii., 8.

33. RANDOLPH, R. L. **A case of blepharochalasis; a variety of dermatolysis.** *Transactions American Ophthalmological Society*, 1916.

34. SHOEMAKER, J. F. **Total absence of eyebrows and eyelashes.** *American Journal of Ophthalmology*, April, 1917.

35. WHITE, D. W. **New operation for canthoplasty with special technic in cases due to trachoma.** *Ophthalmic Record*, March, 1917.

DORE (32, **Involuntary movements of the upper lid**) describes a case of involuntary movements of the upper lid associated with the innervation of other cranial nerves, the Marcus Gunn disease. This condition is called in France "Machoire a clignements" and in England "jaw-winking." Eighty cases of this kind have been described in literature, and a complete bibliography is given at the end of the paper.

SCHOENBERG.

RANDOLPH'S (33, **Blepharochalasis**) case, a girl of 15, had had since birth looseness of the skin and thinness of the connective tissue of the upper lids. There were occasional attacks of itching which provoked scratching and swelling.

ALLING.

SHOEMAKER (34, **Total absence of eyebrows and eyelashes**) observed a young man who, when six years old and after an attack of measles, lost nearly all of his hair. Part of it grew again, but at the age of twenty-two all of it came out, leaving only a few hairs on his body. It is thought that the condition was due to diminished secretion of the ductless glands, probably the thyroid and the gonads, although there were no other symptoms.

ALLING.

WHITE (35, **New operation for canthoplasty**) calls attention to the fact that canthoplasty as usually performed is liable to prove a failure from subsequent contractions, and offers an operation for permanent results. The essential feature is the folding under of the skin of the wound on itself, both above and

below the incision made at the outer canthus. The conjunctiva is then sutured to the wound to cover it. The edges of the lids at the canthus are thus formed of folds of skin and cannot unite.

ALLING.

IX.—LACRIMAL APPARATUS.

36. VERHOEFF, F. H. **A simple and rapid operation for removal of the lacrimal sac.** *Ophthalmic Record*, October, 1916.

VERHOEFF (36, **Simple and rapid removal of the lacrimal sac**) makes his incision in the skin by beginning about 4*mm* from the inner canthus and curving it toward the nose, both above and below. The flap is then dissected up and retracted. He cuts the canthal ligament and comes immediately upon the sac, which is easily removed. Although he has seen no disfigurement as the result of cutting the canthal ligament he usually sutures its ends.

ALLING.

X.—ORBITS, EXOPHTHALMOS, ACCESSORY SINUSES.

37. HOLLOWAY, T. B. **Two cases of orbital growth. (A) Mixed tumor of the lacrimal gland; (B) Small round-cell sarcoma in an infant.** *Transactions American Ophthalmological Society*, 1916.

38. ROLLET. **Orbito-palpebral prothesis for war injuries, made of wax and rubber.** *Archives d'ophtalmologie*, xxxv., 3.

39. VALOIS, G., and ROUVEIX, J. **Prothesis of the orbit.** *Clinique Ophtalmologique*, vii., 8.

40. VEASEY, C. A. **An unusually large osteoma of the frontal, ethmoidal, and sphenoidal sinuses, involving the orbit and the anterior cerebral fossa, presenting no subjective symptoms other than proptosis of the eyeball.** *Transactions American Ophthalmological Society*, 1916.

HOLLOWAY (37, **Orbital tumors**) regards the first case of tumor which he removed as an adenocarcinoma with reversionary infiltration and degenerative changes. The second case was a 14-months infant and the right orbit was eviscerated. Later the left side became affected, metastases developed in the liver, and the child died.

ALLING.

VEASEY'S (40, **Osteoma of the accessory sinuses**) patient had first shown proptosis of the right eye eleven years before and it had gradually increased. She complained only of dull

pain in the eye. The exophthalmos was marked, but the lids could be closed. The optic nerve was slightly paler than normal, but vision was $\frac{6}{6}$. A large tumor could be felt in the upper-inner orbit and X-ray showed that it involved the sinuses and that it extended into the anterior cerebral fossa as a mass about the size of a hen's egg. Operation was thought inadvisable.

ALLING.

ROLLET (38, **Orbito-palpebral prothesis for war injuries, made of wax and rubber**). There are cases with complete destruction of the lids and contents of the orbit so that there can be no question of an autoplastic procedure for prothetic purposes. In such cases Rollet has had Dr. Pont, the chief of the Stomatological Department of Lyon, construct, in one piece, an artificial eye with orbital margins, lids, and ciliæ, which fits exactly in the depression corresponding to the orbit. The prothesis is made of a paste consisting of aquæ 25; glycerine 62; glue 5; pure, white gelatine 20; some yellow is added, to give it the color of the skin. When the paste is ready, it is varnished with a mixture of ether 15, mastic 15, Canada balsam 2.50. The patient makes his own prothesis by heating the paste and pouring it into a mould previously fashioned from his own orbital depression.

Rollet has used this method in seven patients with excellent results, as shown in the reproduced photographs of two of them. SCHOENBERG.

VALOIS and ROUVEIX (39, **Prothesis of the Orbit**) say it is very important to utilize every possible means of ingenuity to give to the soldiers with severe injuries in the region of the face and orbit and destruction of the eyeball the best obtainable prothesis. The authors have gradually worked out a technic of taking proper impressions of the orbital cavity with plaster of paris or preferably with "albâtre." After the moulding is executed they construct a prothesis of hard rubber in the front, which imitates the anterior portion of the eyeball, and of soft, elastic rubber in the posterior portion. This has the advantage of adhering to the tissues and of responding to the slightest movements left behind in the shreds of muscles which have been saved in the process of healing.

SCHOENBERG.

XI.—CONJUNCTIVA.

41. BUTLER, H. **Vernal catarrh and the radium treatment.** *Clinique Ophtalmologique*, vii., 8.

42. COUSIN, G. **New treatment for pterygium.** *Archives d'ophtalmologie*, xxxv., 3.

43. LAMB, H. D. **A case of lipodermoid of the bulbar conjunctiva with accompanying congenital defects.** *American Journal of Ophthalmology*, November, 1916.

44. PATTERSON, J. A. **Tubercle of the conjunctiva.** *Annals of Ophthalmology*, January, 1917.

45. TERSON, A. **On atropine conjunctivitis.** *Clinique Ophtalmologique*, vii., 7.

46. VAN SCHEVENSTEEN, A., Jr. **Remarks on artificial conjunctivitis.** *Ibid.*, vii., 10.

BUTLER (41, **Vernal catarrh and the radium treatment**) considers the radium treatment as specific for vernal catarrh and reviews two cases in support of his belief.

SCHOENBERG.

COUSIN (42, **New treatment for pterygium**) dissects the head of the pterygium from the cornea and cauterizes with the electro-cautery the denuded surface of the cornea and the freed margins of the conjunctiva. The procedure should be repeated three to four times at eight-day intervals.

SCHOENBERG.

The tumor described by LAMB (43, **Lipodermoid of the bulbar conjunctiva**) extended from the cornea into the orbit and was partially removed. It consisted of fibrous tissue, fat, nerve fibers, and a small lacrimal gland. There were no cilia or hair follicles. A coloboma of the iris was present and pupillary membrane. The left testicle had not descended, and there was a congenital defect in the bone of the temporal region.

ALLING.

PATTERSON (44, **Tubercle of the conjunctiva**) saw a case suffering from pulmonary tuberculosis with a large round ulcer on the conjunctival surface of the upper lid, and another, linear in shape, near the edge of the everted lid. The round ulcer was undermined with a gray base. The lymphatic glands were not involved. After cauterization with trichloracetic acid the ulcers healed.

ALLING.

A. TERSON (45, **Atropine conjunctivitis**) recommends euphthalmine as a substitute in patients who cannot tolerate atropine or scopolamine.

SCHOENBERG.

A. VAN SCHEVENSTEEN, Jr. (46, **Remarks on artificial conjunctivitis**) says that he has seen a number of soldiers with artificially induced conjunctivitis. The substance usually employed is ipecac powder, introduced in the lower conjunctival sac. The acute conjunctivitis thus induced is typical: unilateral, of sudden onset, and very violent. The process is limited to the lower conjunctival sac; there is very little secretion. The skin of the lower lid is red and "erysipelatoid" in appearance. The chronic conjunctivitis, kept up by the repeated introduction of ipecac powder, is characterized by a peculiar salmon-like discoloration of the conjunctiva, very scanty secretion, marked thickening of the subconjunctival tissue, very marked tendency of the bulbar conjunctiva to congestion and even inflammation at the slightest manipulation or instillation of zinc or silver-nitrate drops.

Other substances used for the production of artificial conjunctivitis are: podophyllin, jequirity, cantharides, chrysarobin, pepper, soap, tobacco, and dental tartar.

Treatment consists of applying a collodion bandage on the eye for three to four days. SCHOENBERG.

XII.—CORNEA AND SCLERA.

47. CHANCE, B. **Recurrent sarcoma at the limbus treated by electric desiccation.** *Transactions American Ophthalmological Society*, 1916.

48. LAMB, H. D. **Two cases of bacillus pyocyaneus keratitis.** *American Journal of Ophthalmology*, September, 1916.

49. PUSEY, W. A. **Facial pigmented nævus involving the sclera.** *Ophthalmic Record*, December, 1916.

50. SIMON. **Ionotherapeutics (salicyl and lithium) for episcleritis.** *Clin. Ophtal.*, vii., 7.

51. SNELL, A. C. **An epibulbar melanosarcoma of the limbus which completely disappeared under X-ray treatment.** *Transactions American Ophthalmological Society*, 1916.

52. THEOBALD, S. **Observations upon herpes corneæ febrilis with reference especially to ætiology.** *Ibid.*, 1916.

LAMB (48, **Bacillus pyocyaneus keratitis**) saw two cases of extensive destruction of the cornea caused by rapid erosion of

ulcers. It would seem that the ulcers due to pyocyaneus have a distinct pathology. They are characterized by rapid progress over the surface and into the substantia propria—from three to seven days being sufficient to destroy nearly the whole cornea. The process is self-terminating and, in throwing off the slough, leaves a clean transparent base. There is no specific treatment.

ALLING.

THEOBALD (52, **Herpes corneæ febrilis**) thinks that the qualifying term febrilis is not well chosen for the type of case which shows unilateral diffuse, irregular, or dendritic corneal opacities not affecting the substantia propria and associated with hypesthesia. He believes that the lesion is in the ciliary ganglion in spite of the fact that this ganglion is supposed to be purely efferent and not to contain sensory fibers. There is some evidence, however, in favor of its double nature. He uses holocaine, atropine, and dionin, with quinine and potassic iodide internally.

ALLING.

The tumor when seen by CHANCE (47, **Recurrent sarcoma at the limbus treated by electric desiccation**) was about the size of a bean and projected through the palpebral fissure. It arose from the sclerocorneal junction and extended well on to the cornea. It was excised three times but recurred. The tumor was then desiccated by heat in the form of electric flame produced by a high-frequency current from a static machine. After six weeks the area looked healthy and the eye was quiet. Another case of pigmented pedunculated sarcoma which recurred was also treated successfully by the same method. Desiccation possesses the advantage over X-ray in that its action can be seen directly and confined to definite areas.

ALLING.

The tumor treated by SNELL (51, **An epibulbar melanosarcoma of the limbus which completely disappeared under X-ray treatment**) extended about two thirds about the periphery and involved the cornea nearly to the center. It was divided into eight distinct nodules and had been excised without success. Twelve treatments with X-ray were given during three months after which the mass had disappeared except

for a few small islands of brown pigment. Seven months later the eye was reported to be free from trouble.

ALLING.

PUSEY (49, **Facial pigmented nævus involving the sclera**) describes a case of extensive nævus of the skin of the face which also involved the sclera except for a small area on the inner side.

ALLING.

SIMON (50, **Ionotherapeutics (salicyl and lithium) for episcleritis**) has succeeded in clearing up episcleritis in five patients in a remarkably short time by the method of ionization. He has constructed an eye-cup, connected with the pole of a galvanic battery. The cup contains a 1% solution of lithium salicylate and the current used is never stronger than 1 m. a. for 6 minutes for the salicyl-ion and 1–2 m. a. for 3 minutes for the lithium-ion. The application should be made every two days.

SCHOENBERG.

XIII.—IRIS AND PUPILS.

53. BROWN, E. V. L., and IRONS, E. E. **The ætiology of iritis.** *Transactions American Ophthalmological Society*, 1916.

54. RING, G. O. **Concerning coloboma of the iris in association with congenital cataract.** *Ophthalmic Record*, March, 1917.

55. TERRIEN, F., and MAWAS, J. **Extraction of persistent pupillary membrane.** *Archives d'ophtalmologie*, XXXV., 4.

BROWN and IRONS (53, **Ætiology of iritis**) present the results of exhaustive studies of 100 patients with a view to determining the cause of iritis. In a surprising number they found two or more possible causes associated. Thirty-nine cases gave evidence of syphilitic infection but in only 23 did this cause seem to be the occasion of the iritis. They think therefore that the widely accepted notion that one half the cases are syphilitic needs revision. Infection from the teeth is very common, but care must be taken in ascribing the iritis to this cause. In 15 cases the iritis was undoubtedly due to some other cause, with the dental infection secondary. In 9 patients they were unable to determine the relative importance of the different sources of infection. The tonsils were clearly the source in 16 cases. Gonorrhœa was believed to

be the cause in 9 patients. In 8 tuberculosis was assigned and in 3 the sinuses.

ALLING.

RING (54, **Coloboma of the iris with congenital cataract**) presents the case of a mother and two children who showed microphthalmos, coloboma of the iris, and posterior polar cataracts. It is suggested that the fact that the grandmother of the children had suffered from insufficient food and ill treatment might have had some influence in production of the deformities.

ALLING.

TERRIEN and MAWAS (55, **Extraction of persistent pupillary membrane**) report the successful extraction of a persistent pupillary membrane. The vision before operation was $\frac{1}{10}$; after the operation normal. The method of operation consisted of first separating with a discission knife the lower "tendons" of the membrane, a few days later the lateral "tendons" were incised, and after the anterior chamber was re-formed an iris forceps was introduced through a keratome incision in the limbus and the membrane extracted.

SCHOENBERG.

XIV.—LENS.

56. AUBINEAU, E. **Cataract and aphakia from the military point of view.** *Clinique Ophtalmologique*, vii., 4.

57. BRUNS, H. D. **The ambulant treatment of cataract extraction; with a note on postoperative delirium and striped keratitis.** *Transactions American Ophthalmological Society*, 1916.

58. DE GUILLEMA, SIMON. **The treatment of cataract by iodionization.** *Ibid.*, vii., 10.

59. JOCQS. **Pathogenesis of cataract.** *Clinique Ophtalmologique*, vii., 7.

60. ROLLET and VELTER. **Indications for the extraction of traumatic cataract in wounded soldiers.** *Ibid.*, vii., 6.

61. VERNEY, L. **A contribution to the question of spontaneous resorption of cataracts.** *Ibid.*, vii., 4.

62. WHEELER, J. M. **A study of hemorrhage into the anterior chamber subsequent to cataract operations for hard cataract.** *Transactions American Ophthalmological Society*, 1916.

JOCQS (59, **Pathogenesis of cataract**) makes a critical review of the theories advanced by a number of authors who have studied the subject of the pathogenesis of cataract—mostly

from the experimental point of view. He concludes that the main factors to be considered in the causation of cataract are: (1) A disturbance of the excretory mechanism of the organism. (2) A damaging of the epithelial layer of the anterior portion of the capsule of the lens by a toxic substance retained in the organism.

SCHOENBERG.

DE GUILLEMA (58, **Treatment of cataract by iodionization**) reports the history of a patient 69 years old with a bilateral senile nuclear cataract, in whom the vision remained stationary for several months because, as he believes, of the ionization treatment administered.

SCHOENBERG.

E. AUBINEAU (56, **Cataract and aphakia from the military point of view**). According to military regulations a cataractous eye is a reason for transferring a soldier from active into auxiliary service. The author advises operation of every operable cataract for the reason that an aphakic eye has the advantage of a greater field of vision and also of the possibility of getting a good deal of vision with correction in case something happens to the other eye. The liability of the state is much less towards aphakics than towards soldiers with non-operated cataractous eyes, and this should be another reason for operation.

SCHOENBERG.

ROLLET and VELTER (60, **Indications for the extraction of traumatic cataract in wounded soldiers**) found that out of 25,000 eye cases 237 had a traumatic cataract and 123 were operated upon. They classify these cataracts in two groups: with and without wounds of the eyeball.

Regarding the traumatic cataracts without wounds of the eyeball, the indications for operation are put down as follows:

1. Luxation of the lens or its complete opacification.
2. Hypertension not responding to myotics.
3. Very poor vision in the other eye.

The operation is contraindicated if the other tissues of the eye are severely damaged. Of 40 traumatic cataracts without an external injury of the eyeball, 14 were operated upon. Traumatic cataracts with wounds of the eyeball are more frequent (197 out of 237 cases). The first thing to do is to

extract the foreign body, if present. The cataract is removed at the same time, if possible; if not, several weeks later when the reaction has entirely subsided. Of 20 cases with traumatic cataract and intraocular foreign body, 6 had the lens extracted some time after the removal of the foreign body, 12 had only the lens extracted, and in 2, the foreign body in the lens was extracted. The results were quite satisfactory, vision ranging between $\frac{1}{4}$ and $\frac{1}{50}$.

Regarding the indications for the extraction of traumatic cataracts without foreign bodies in the eyeball, which are about five times more frequent than those with intraocular foreign bodies, the authors formulate them as follows:

1. Immediate extraction, if the wound is large, and the capsule is torn.

2. If there is hypertension which does not respond to myotics.

Wounds extending to the posterior half of the eyeball with damage of the choroid and retina, with symptoms of infection like iritis and iridocyclitis with hypotension, are distinct contraindications for operative treatment. The authors extracted the lens in 89 out of 166 of this group of cases and obtained $\frac{1}{4}$ to $\frac{1}{50}$ vision. It is very interesting that in spite of the hurry and untoward circumstances under which the operations had to be performed, none of the patients showed any signs of postoperative infections.

SCHOENBERG.

BURNS (57, **Ambulant after-treatment of cataract extraction**) sends his patients home after the operation, leaving the other eye open. They return to the clinic daily for dressings. Before beginning this method he had made 371 extractions with the usual after-treatment and had ten per cent. failures. Under this treatment he has had 232 cases and seven per cent. failures.

Postoperative delirium, which he attributes to strange surroundings and confinement, does not occur when the patients are sent home.

ALLING.

In an analysis of 2123 operations at the N. Y. Eye and Ear Infirmary, WHEELER (62, **Hemorrhage into the anterior chamber after cataract extraction**) found that this accident

occurred in 4.57%. It seems that hemorrhages are more liable to appear in the middle-aged rather than in the young or very old. It occurred from one to twenty-eight days after the operation. Diabetes and albuminuria predispose the hemorrhage and it is most common after the combined operation and least after the preliminary iridectomy.

ALLING.

L. VERNEY (61, **A contribution to the question of spontaneous resorption of cataracts**) attempted to remove a cataract from a patient 56 years of age. There were numerous posterior synechiæ. At the operation only the capsule and some fluid were found. No nucleus. The same patient had been operated for a cataract by another ophthalmologist some twenty years before and the same condition found then. Verney reviews the literature on the subject and expresses his belief that in eyes with pathological conditions like chronic iritis, glaucoma, chorioretinitis, etc., sometimes, toxic substances penetrate the capsule of the cataractous lens, the fibres liquefy, and the nucleus disappears. At a certain period the capsule may burst and its contents pass into the aqueous. By nature's method of spontaneous resorption of the cataract the vision may return in eyes which had been blind for many years.

SCHOENBERG.